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RADICAL LOBECTOMY

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THIS REPORT defines a radical lobectomy as an operation in which one or two lobes of an entire lung are excised in a block dissection with certain of their regional hilar and mediastinal lymphatics.

The technical steps and clinical implications of the procedure to be discussed are based upon an experience with 48 radical lobectomies performed on the Thoracic Surgical Service of Memorial Center.

TECHNIQUE

In the description of the technique to follow, emphasis is placed upon the method of lymph node dissection. The ligation and division of the tributary vessels and the bronchi to the various lobes are not included.

The first step of each radical lobectomy ideally proceeds from the most distal area of potential spread and works back toward the hilum and the tumor. However, this cannot always be achieved because adhesions and many other conditions in the thorax can obliterate clear anatomic planes. Instead of starting with the lymphatic dissection it may be necessary to begin at the hilum of the lobes by entering their fissures and proceeding distally, or by using a combination of these techniques. Another technical variant may be indicated when the tumor invades chest wall. It may be difficult to approach the hilum until this extension or peripheral fixation is mobilized to rest free, attached to the lung.

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To approach either lung, the patient is placed in a true lateral position. This is preferred to the supine or prone position, for it provides an optimum exposure for mediastinal lymph node dissection. The chest is entered through a long parascapular incision and the bed of the fifth or sixth rib. The pathologic setting is assessed, and if a radical lobectomy seems indicated and feasible and the patient's general condition is satisfactory, the procedure is carried out.

A Right Upper Lobe Radical Lobectomy (Fig. 1).—This procedure begins with the incision of the superior mediastinal pleura, from the apex of the chest to the azygos vein arch just anterior to the vagus nerve. Superiorly, a short horizontal limb is used to facilitate exposure of the subclavian artery and right recurrent laryngeal nerve.

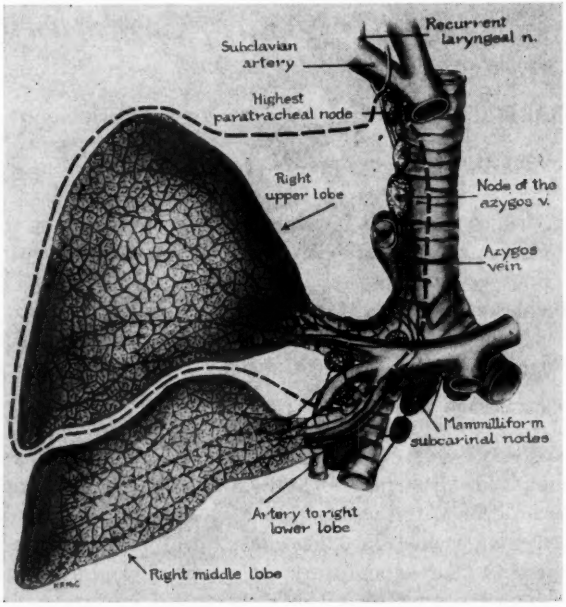

Fig. 1.—Right upper lobe radical lobectomy, anterior view. Limit of excision diagrammed by interrupted line. Note inclusion of nodes along the intermediate bronchus and the important azygos vein node. Stump of right lower lobe bronchus shown; lobe omitted for clarity.

For the paratracheal dissection, the anterior pleural flap is elevated to the anterolateral surface of the superior vena cava, the phrenic nerve being displaced with it.* The posterior flap is mobilized as far as the vertebral bodies, leaving the vagus nerve uncovered. These flaps are retracted by the weight of the hemostats fastened to sutures attached to their free edges.

The azygos vein is transected between ties and ligatures, and the anterior limb lifted forward, thereby elevating the superior vena cava to some extent.

*Although there are lymph nodes lying in the periphrenic areas as well as anterior to the superior vena cava and innominate veins, these nodes have not been included, for they lie beyond the arbitrary limits of the dissection as suggested by the superior vena cava. It was also thought that the route of lymphatic spread on the right side was predominantly paratracheal and retrocaval to the jugulo-subclavian junction.

The areolar tissue is cleared laterally and posteriorly from the superior vena cava from the apex of the chest to an area just beneath the azygos vein junction with it. The upper thoracic portion of the vagus nerve is mobilized and the take-off of the right recurrent laryngeal branch is identified. The vagus is transected just distal to that point. This facilitates a clearer dissection of the paratracheal areolar tissue.

The apex of the dissection is the inferior border of the subclavian artery where a tongue of areolar, node-bearing tissue is isolated and divided.

Next, attention is directed to the trachea. The fine connective tissue on its right lateral and anterior surfaces is found to separate readily, allowing the whole packet of tissue anterior to the trachea, including the areolar tissue posterior to the trachea and on the esophagus, to be swept downward off the innominate artery and aortic arch to the level of the pulmonary artery. This can usually be done with division of only a few small vessels. This packet of tissue represents the right paratracheal lymph node chain. To maintain the integrity of this block of lymphatics, the dissection must be carried immediately adjacent to the large vessels and the trachea.* Included in this dissection is the particularly important, constant, large node of the azygos arch lying just above the pulmonary artery. It is the one most frequently involved in cancers of the right upper lobe. This node is mobilized as the dense connective tissue superior to the pulmonary artery and behind the superior vena cava is incised. This paratracheal node packet remains attached to the lung by peribronchial lymphatics and areolar tissue. As each packet of nodes is mobilized, it should be tacked to a convenient point on the visceral pleura to keep them from obscuring the later dissection.

An incision is then made in the interlobar fissure separating the upper lobe from the middle and lower lobes and carried down to the origin of the right upper lobe bronchus. A group of nodes can be found surrounding the main-stem bronchus. The posterior nodes of this group are found near the anterior edge of the esophagus on the right. On the left, they are less frequently found. These are dissected toward the specimen.

Lymph nodes are placed along the broncho-arterial trees of each lobe, and it is important to note that the principal lymph node draining one lobe group may rest against the bronchus of an adjacent lobe. The continuation of the right main-stem bronchus to form the middle and lower lobe bronchi (intermediate bronchus) can be traced and it is usually feasible, and indeed of decided importance, to include the nodes that present themselves adjacent to these structures. These are nodes that Borrie described as being in the "sump" position,[2] and usually represent the lowermost site of spread from right upper lobe lesions. The division of the upper lobe bronchus can be helpful in providing a more thorough dissection of the retrocaval and peribronchial nodes.

*In order to accelerate the dissection of the small vessels in the mediastinum, silver brain clips (McKenzie) are used for hemostasis instead of ligatures.

It is necessary, on occasion, in the treatment of lesions of the right upper lobe, to include the middle lobe as part of the block dissection. Such a bi-lobectomy is usually performed for the following three reasons: (1) when there is a partial to almost total symphysis of these two lobes, (2) when the tumor transgresses one lobe and is fixed to or invades the other, and (3) when it is thought that a more complete dissection of the "sump" area along the intermediate bronchus can be obtained.

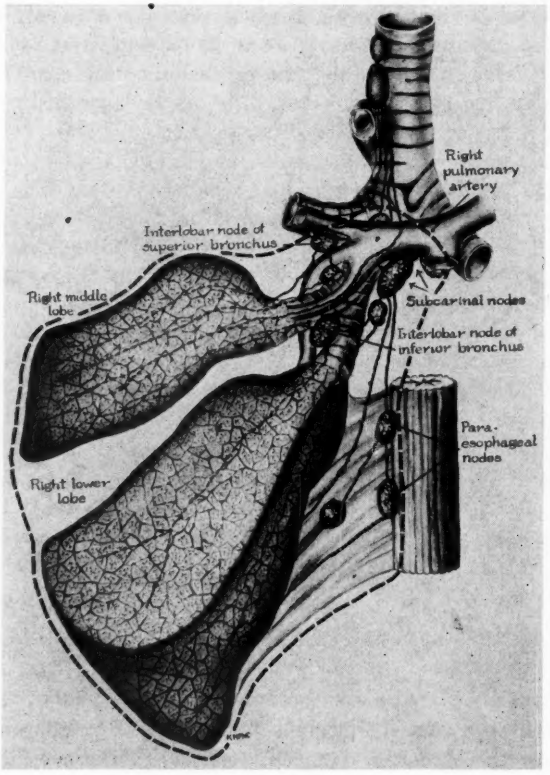

Fig. 2.—Right middle and lower lobe radical lobectomy (anterior view) showing line of excision. Note inclusion of pre-esophageal, subcarinal, and interbronchial nodes. Paratracheal chain may be removed as a separate specimen.

A Middle Lobe Radical Lobectomy.—It is improbable that the middle lobe would be removed by itself if cancer were suspected, except in the most unusual circumstances such as in very poor-risk patients. Tumors within it are almost always excised in combination with a right upper lobe radical lobectomy. To such a bi-lobectomy are added not only all the nodes in the "sump" area but as many as are available about the origin of the adjacent lower lobe bronchus as well.

At all times the indications for one operation or another are subject to flexibility and depend to a large extent upon the intrathoracic setting. For example, should the tumor extend between the middle and lower lobes, a lower lobe radical lobectomy en bloc with the middle lobe may be performed, leaving the right upper lobe intact. However, the lymph node dissection of the upper mediastinal and paratracheal nodes, including the azygos node, may then be done separately.

A Right Lower Lobe Radical Lobectomy.—This dissection usually includes the middle lobe. It begins at the diaphragm, with a division between ligatures of

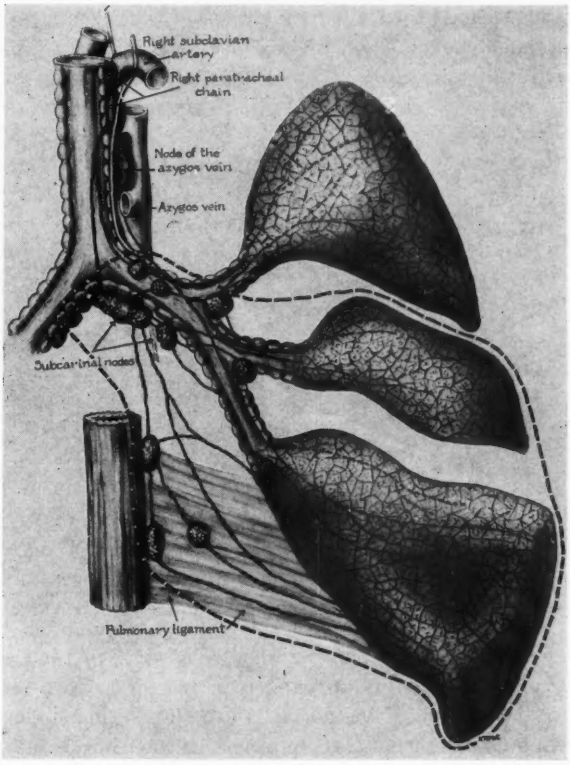

Fig. 3.—Right middle and lower lobe radical lobectomy (posterior view).

the inferior pulmonary ligament[14] (Figs. 2 and 3). The esophagus is identified and the areolar and lymphatic tissue along its anterolateral surface is elevated upward as far as the inferior pulmonary vein and tacked to the lower lobe. Steady, gentle traction on the lower lobe in an anterior direction will expose the subcarinal node packet. At this level, this will become more available if the esophagus is retracted sharply posteriorly. The inferior and medial edge of the subcarinal packet is usually easily dissected from the adjacent pericardium. It is

not believed necessary, as some authors advocate,[3] to excise routinely a portion of the pericardium with this packet, for only the most filamentous areolar connective tissue lies between this lymph node group and the pericardium. By entering the plane adjacent to the posterior edges of the *left* or contralateral bronchial cartilages, simple and practically avascular mobilization of this nodal packet is effected.

Dissection is then carried to the main-stem bronchus, exposing its posterior surface. The origin of the right upper lobe bronchus is identified and the nodes are taken from the intermediate bronchus and reflected toward the specimen. After identifying the right middle and lower lobe bronchi, their transection at this time will permit the inclusion of the more cryptically placed nodes about the origin of the right middle lobe bronchus. The division of the tributary arteries and veins to these two lobes can then be made.

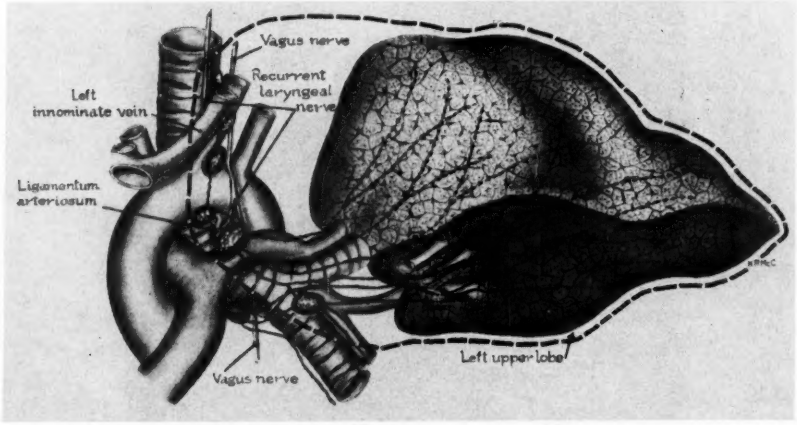

Fig. 4.—Left upper radical lobectomy. Interrupted line marks limits of dissection. Note inclusion of interbronchial lymph node ("sump" position) between upper and lower lobe bronchi. Subcarinal nodes removed as a separate packet.

A Left Upper Lobe Radical Lobectomy (Fig. 4).—Incision is made into the mediastinal pleura, beginning at the apex of the chest and proceeding along the vagus nerve to the arch of the aorta. Its posterior limb follows the aortic arch to the level of the bronchus. Its anterior limb extends 1 to 2 cm. anterior to, and inferiorly along, the course of the phrenic nerve to the inferior pulmonary ligament to join the posterior incision.

Flaps are elevated and retracted. The phrenic nerve is fully liberated throughout most of its upper mediastinal course so that it may be retracted anteriorly and posteriorly by a Penrose drain as the dissection warrants.

The apex of the anterior mediastinal dissection is at the under surface of the left innominate vein at a point where it crosses the left subclavian artery. The areolar, node-bearing tissue is dissected off the vein, and several small branches must be separately identified and divided. The dissection proceeds

anteriorly beneath the innominate vein as far as its entrance into the superior vena cava, dividing the highest intercostal and the thymic branches along the way. This is the anterior limit of the dissection.

The posterior limit of the dissection is the subclavian artery. The packet of tissue is swept down off the subclavian, common carotid, and innominate arteries and the aortic arch. The vagus nerve is carefully cleaned and, in particular, the large lymph node present in the space between the common carotid and subclavian arteries is included in the specimen.

Beneath the aortic arch, the node of the ligamentum arteriosum* and that of the loop of the left recurrent nerve are freed. Lying between the superior border of the pulmonary artery and the anterior surface of the left main bronchus and extending beneath the aorta is a packet of nodes which must be dissected down toward the specimen. All of these are included with the previously dissected mediastinal packet of tissue and tacked onto the lung surface.

The fissure between the upper and lower lobes is then divided and an approach made to the hilum. The incision made into the visceral pleura is curved anteriorly and posteriorly to meet the incision made previously in the mediastinal pleura. Starting with the bronchus to the left lower lobe, the important lymph nodes that are readily available in the "sump" position are swept upward between the lower lobe bronchus and its corresponding artery up to the take-off of the upper lobe bronchus. This last structure is isolated while dissecting the lymphatics toward the lung substance. By division of the artery to the left upper lobe, the dissection of nodes from the superior border of the left main-stem bronchus is facilitated and all these are included with those from the subaortic and anterior mediastinal dissections. The subcarinal nodes are occasionally included as a separate packet. This practice becomes more routine in light of the presence of metastases within them on several occasions.

A Left Lower Lobe Radical Lobectomy (Figs. 5 and 6).—This procedure is similar to that on the right, including the pre-esophageal, subcarinal, and the peribronchial nodes ("sump" position) located along the left main-stem bronchus and immediately available around the left upper lobe bronchus. As on the right side, the left phrenic nerve is optionally crushed if a lower lobe is removed. The lingular portion of the left upper lobe is not included although, as has been noted, the right middle lobe is taken as part of a right lower lobe radical lobectomy.

Many patients who have a radical lobectomy performed are actual or potential pulmonary cripples, so that every effort is made to prevent compensatory emphysema of the lower lobes when an upper lobe lobectomy is performed. The space previously occupied by the upper lobes has been filled as follows: the parietal pleura occupying the dome and walls of the thorax is mobilized by extrapleural dissection so that it collapses on the superior surface of the lower lobe. An Ivalon sponge is then inserted in this extrapleural space, after it has been autoclaved in saline, tailored to size and shape, and

*The ligamentum arteriosum has been divided in some instances to permit a more thorough node dissection beneath the arch of the aorta.

rinsed in 200 to 300 c.c. of a saline solution containing one million units of penicillin and one gram of streptomycin. Under-water drainage tubes are inserted separately, both into the pleural cavity and into this extrapleural space. The drainage of the pleural cavity is ended about the third or fourth day, and the extrapleural space about a day or two later. The extrapleural insertion of these Ivalon sponges has been performed in 8 instances without any morbidity or mortality. The longest period that one has been in place is 7 years (Fig. 7).

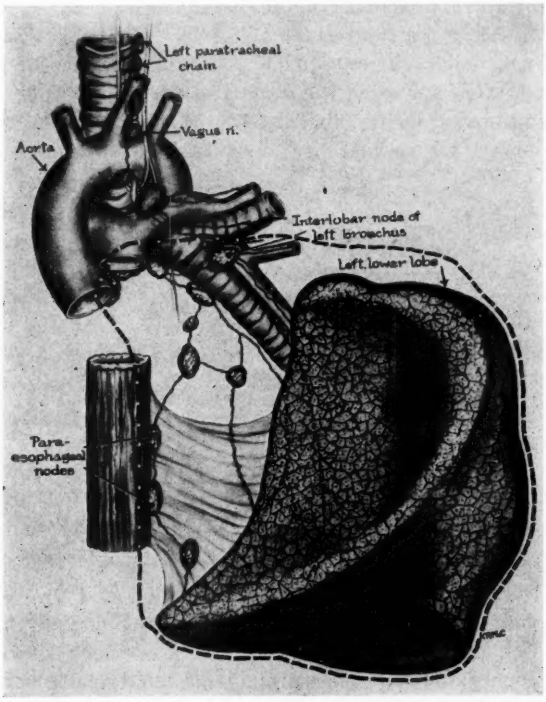

Fig. 5.—Left lower lobe radical lobectomy (anterior view). Interrupted line indicates limits of dissection. Note inclusion of the interbronchial node ("sump" position) as well as the subcarinal paraesophageal groups.

With the same principle in mind, there is the option of crushing the phrenic nerve after the excision of the middle and lower lobes. This causes an elevation of the leaf of the diaphragm which will fill to a large extent the space left by the lobectomies. The virtue of this, in preventing excessive compensatory emphysema of the right upper lobe as well as reducing dead space, must be balanced against the paradoxical respirations and attendant mediastinal swing that will occur.

INDICATIONS

In the following discussion it must be assumed that the pulmonary lesion is apparently contained within the confines of a lobe so that the transection

of the lobar bronchus and vessels will provide a reasonable margin of uninvolved tissue. Such a determination can, in part, be made preoperatively by bronchoscopic visualization of the major bronchi. At the time of thoracotomy, palpation and limited dissection about the hilum will also help to decide the feasibility of a lobectomy. At best this is only a gross estimate of tumor boundaries and and can often be clouded by adjacent inflammation, fibrosis, and healed calcified tuberculous lymph nodes.

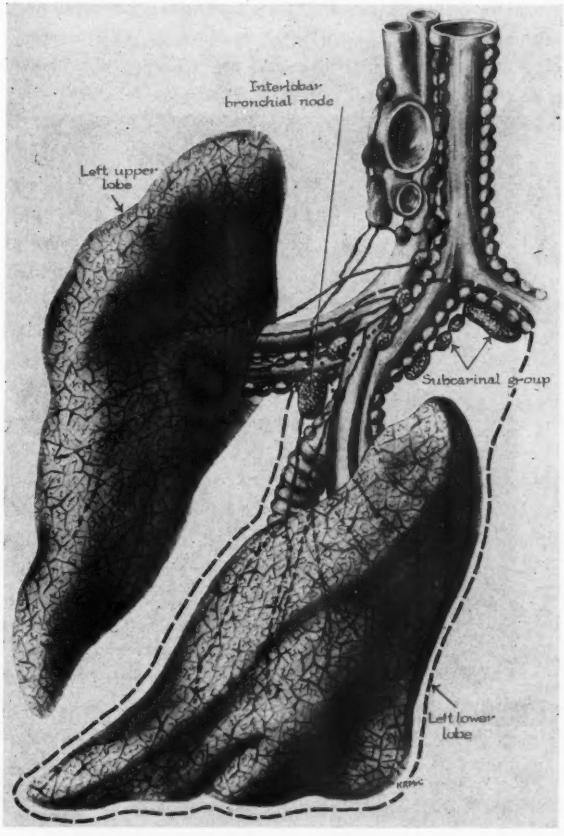

Fig. 6.—Left lower lobe radical lobectomy (posterior view).

There are two major indications for a radical lobectomy. These are for the treatment of (1) a primary cancer of the lung under certain circumstances, and (2) a lung tumor the nature of which is ambiguous.

Primary Cancer of the Lung.—The most important indication is for the excision of a peripherally placed cancer of the lung in a patient who, it is deemed, cannot withstand a radical pneumonectomy. The interpretation of what is ordinarily called "a poor-risk patient" varies from institution to institution and surgeon to surgeon. It is necessarily a flexible term because it is

an appraisal of risk based upon not only fairly general measurements, but also subject to re-interpretation in the operating room. In other words, the patient who may be considered a good operative risk from the point of view of cardiorespiratory physiology may on occasion demonstrate bizarre physiologic responses to endotracheal intubation and the opening of his chest which would tend to modify the extent of the contemplated procedure. At this institution, reliance is placed upon a few arbitrary preoperative findings which might, either by themselves or in association with other abnormalities, influence the magnitude of the operation. For the most part, patients have been 75 years and over. They may have evidence of marked emphysema both by physical exam-

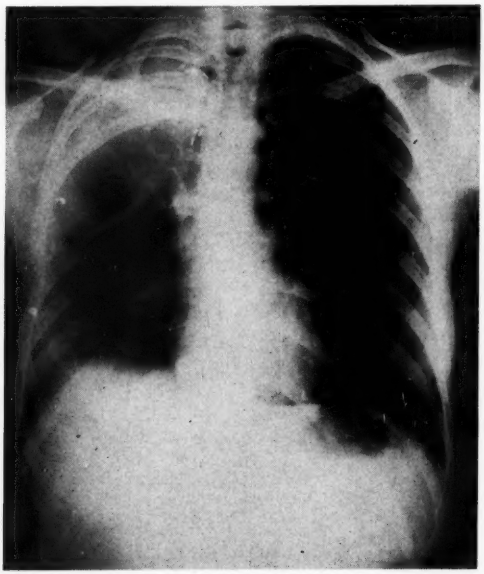

Fig. 7.—Postoperative roentgenogram of patient in Fig. 8 showing Ivalon sponge in right upper retropleural space and McKenzie clips in place along the paratracheal area. Note how little tracheal shift or diaphragmatic rise has occurred after a right upper and middle lobe radical lobectomy.

ination and maximum breathing capacity estimates, or they may have extensive myocardial disease. One condition that carries great weight in influencing decision in this regard is the history and finding of bronchial asthma.

We do not at present elect to do a radical lobectomy for cancer of the lung as the treatment of choice. However, there is suggestive evidence that a right upper lobe radical lobectomy might become the preferred procedure for cancers grossly confined to the right upper lobe. Even now, some surgeons[9, 11] choose a simple lobectomy for these peripherally placed cancers, regardless of the clinical condition of the patient. Their experience will provide a base line of comparison for the value of adding the more extensive lymph node dissection.

Theoretically, a radical lobectomy might be less advantageous for primary lung cancers in the right middle and right lower lobes, for the usual lymphatic currents would tend to favor metastases at locations beyond the usual limits of a block dissection of a right middle and lower lobe radical lobectomy (although these may be excised as a separate specimen). The same is true for cancers in the left lower lobe. Here the possibility of metastases extending first to the subcarinal and then contralaterally to the unavailable right paratracheal lymph nodes makes a more complete block extirpation difficult. Lesions in the left upper lobe (particularly the superior portion) might have a more favorable outlook than those in the left lower lobe because their lymph node metastases tend to remain ipsilateral. However, they too have a tendency to extend to the lymph nodes in the right by way of the subcarinal and anterior mediastinal channels.

A Lung Tumor Whose Nature is Ambiguous.—There are 2 clinical settings in which ambiguity surrounds the nature of a lung tumor: (a) A cancer primary at another site is associated with a solitary lung shadow. This lung shadow may represent either a separate primary lung cancer or a solitary metastasis.[6] (b) A mass in the lung resembles cancer, but the diagnosis cannot be established with absolute finality, either before or during the operation.

a. In our experience, we have been impressed by the frequency with which multiple cancers, one of which is primary in the lung, have coexisted. At Memorial Center we have collected 165 such cases. In the same period of time (1926 to 1958), there have been 46 solitary metastases removed. It is not safe, therefore, to presume that a solitary lung shadow, either synchronous or metachronous with a cancer elsewhere in the body, is a metastasis. In fact, these figures suggest that it might be 4 to 1 in favor of its being a separate primary cancer.* In either event, whether separate primary or metastasis, we know that local lymphatic extension can occur. This does not need documentation in the case of primary cancer of the lung, but in 9 metastases to the lung removed by radical lobectomy, there were 6 instances in which daughter metastases existed in the hilar and mediastinal lymphatics (Table I). In other words, a metastasis from another site deposited within the lung parenchyma can behave as if it had its origin there and its metasteses can in turn proceed by lymphatic or venous routes.

b. As is so often the case, an area of inflammation will frequently be present in association with cancer of the lung. This condition usually follows an obstruction of one of the bronchioles, which can produce distal atelectasis, pneumonia, and abscesses. It is not always possible, by preoperative cytologic studies or by biopsy at the time of thoracotomy, to obtain a diagnosis either of a benign or a malignant process. In other words, it is not infrequent at

*In my article, The Ambiguity of a Solitary Lung Shadow in the Presence of a Primary Cancer Elsewhere *in* Medical Science, October, 1958, a rule of thumb was given as an aid for the diagnostic dilemma: "If the patient has a squamous carcinoma elsewhere in the body, the lung lesion is usually a separate primary and predominantly a squamous carcinoma as well. If the patient has a soft part or skeletal sarcoma or melanoma elsewhere, the solitary lung lesion is almost always a metastasis from these. If there is an adenocarcinoma elsewhere, there is about a 50 per cent chance of the solitary shadow being a primary lung cancer." This probability may be higher if there is a lymphoma present.

TABLE I. METASTASES TO LUNG

Number of cases		9
Positive extrapulmonic nodes		
Adenocarcinoma, breast	2	
Adenocarcinoma, rectum	1	
Teratocarcinoma, ovary	1	
Osteogenic sarcoma, femur	1	
Clear cell carcinoma, kidney	1	
	6	
Negative extrapulmonic nodes		
Fibrosarcoma, thigh	1	
Melanoma, skin of axilla	1	
Rhabdomyosarcoma, thigh	1	
	3	

the time of exploratory thoracotomy that repeated biopsies and frozen section analysis of suspicious lung areas can be reported as showing chronic inflammation. This leaves in doubt whether these sections are truly representative, or whether there is a more cryptically placed cancer.* In this equivocal setting it is probably wisest to assume cancer to be present.† As a compromise, therefore, a radical lobectomy is performed. This operation, while fulfilling the desirability of removing the tumefaction in the lung and many of its regional lymphatics, does not subject the patient to the increased hazard of a pneumonectomy.

RESULTS

There were 48 radical lobectomies performed in the 7-year period from March, 1951, to March, 1958. The first was performed in March, 1951. The patient, a woman, had had an adenocarcinoma of the rectum removed 9 years previously, and a solitary shadow appeared in the left upper lobe. She underwent a left upper lobe radical lobectomy and the pathologic report showed a "metastatic adenocarcinoma consistent with rectal origin." There were two positive mediastinal lymph nodes found in the supra-aortic packet (anterior mediastinal chain) that was part of the dissection. The patient is without evidence of disease 8 years and 4 months later (July, 1959).

In this series there were no postoperative complications other than those which caused 5 postoperative deaths: one from gastrointestinal hemorrhage, one caused by overwhelming infection, another by a myocardial infaret, a fourth by hemorrhage in the operating room with cardiac failure, and one from an undetermined cause. This last occurred in a woman who had had a right

*There may be some question as to the advisability of a biopsy by aspiration or incision of a suspected lung tumor. This applies also to the excision of individual lymph nodes from the mediastinum. Spjut[39] found malignant cells in pleural cavity washings of 13 of 28 patients who had lobectomy, pneumonectomy, or exploratory thoracotomy along with excision of tissue for frozen section. Only 3 positive washings were obtained from 21 patients who had resection not preceded by biopsy. Although such free-floating cells are of apparently frequent occurrence, it is unusual to find new actual implants that could be directly attributed to them. However, one wonders if the manipulation and traumatization attendant upon these biopsies does not promote hematogenous dissemination. In addition, if the suspected lesion is active tuberculosis, incisional biopsy can cause spillage of the tubercle bacilli into the pleura.

†Just recently, a lesion removed by wedge excision and appearing grossly and by frozen section as a tuberculoma was found on paraffin section to have terminal bronchiolar carcinoma at its periphery. This finding of cancer in juxtaposition with healed tuberculosis is not uncommon.

middle and upper lobe radical lobectomy performed in July, 1954, for an adenocarcinoma of the lung, and the last operation was a left upper radical lobe lobectomy in December, 1955, for terminal bronchiolar carcinoma which was probably a separate primary cancer.

This mortality rate is high. There is no reason, however, to attribute any of the deaths to the more elaborate lymphatic dissection, a contention that is supported by the fact that there have been no postoperative complications or mortality in the last 13 radical lobectomy patients. Over one half of these operations were performed by the surgical residents under supervision as one of their earliest thoracic procedures.

Primary Lung Cancer.—There were thirty-four primary lung cancers removed. The histologic types most commonly found (Table II) were present, except oat cell carcinoma. In this group, 11 had lymph nodes containing cancer beyond the limits of a simple lobectomy.

TABLE II. PRIMARY LUNG CANCER

Number of cases		34
Contents of lymph nodes not mentioned		2
Determinable cases		32
Positive extrapulmonic nodes		
Epidermoid	3	
Terminal bronchiolar	4	
Anaplastic	1	
Adenosquamous	1	
Adenocarcinoma	2	
	11/32	= 34%
Negative extrapulmonic nodes		
Epidermoid	7	
Terminal bronchiolar	5	
Adenocarcinoma	4	
Muco-epidermoid	3	
Anaplastic	1	
Pleomorphic reticulum cell sarcoma	1	
	21/32	= 66%

Five or more years ago, 4 patients with primary lung cancer had a radical lobectomy (Table III). Two of these had negative hilar and mediastinal lymph nodes. One died 27 days postoperatively of a myocardial infarct, and the other is alive and well having had a terminal bronchiolar carcinoma of the right lower lobe. Two patients had positive extrapulmonic lymph nodes; one, who is alive and well 7 years later, had an adenosquamous carcinoma of the right upper lobe, one died of disease 13 months postoperatively.

Three to 5 years ago, 18 patients were operated upon. In this group there were 5 patients who had extrapulmonic lymph nodes containing metastases. Of these 5, 2 died postoperatively, 2 died of disease 18 and 26 months postoperatively, and one was without evidence of disease and lost to follow-up 12 months postoperatively. Eleven patients had negative hilar and mediastinal

TABLE III. 5-YEAR SURVIVALS OF PATIENTS AFTER RADICAL LOBECTOMY FOR CANCER

Patients operated on *5 or more years* ago		8
Primary lung cancer	4	
Metastases to lung	4	
Patients with *positive* mediastinal nodes		5
Alive and well, 5 or more years		3
Conditions of 5-year survivors with *positive* mediastinal nodes:		
1 primary adenosquamous carcinoma of lung		
1 metastatic rectal carcinoma		
1 metastatic teratocarcinoma of ovary		
1 osteogenic sarcoma (died of disease 7 years after radical lobectomy)		
Patients with *negative* mediastinal nodes		2
Died postoperatively		1
No evidence of disease		1

lymph nodes. Six of these are alive and well 3 to 5 years later. Three died of disease 17, 24, and 30 months postoperatively; one died 23 days postoperatively. In 2 patients the state of the mediastinal lymph nodes was not mentioned. One of these patients died of disease 6 months postoperatively, and the other was alive and well over 3 years later.

Metastases.—Of the forty-eight radical lobectomies, nine were performed for metastases to the lung. There was no postoperative morbidity or mortality in this group. The sites of origin are shown in Table I.

Of these 9 patients, 6 had hilar and mediastinal lymph nodes containing cancer beyond the limits of excision of a simple lobectomy.

Five or more years ago, 4 patients in this group had excision of metastases to the lung (Table III). Three of them had positive mediastinal lymph nodes. These included metastases from rectal cancer, a teratocarcinoma of the ovary (Fig. 8), and 1 osteogenic sarcoma. The first 2 are alive and well, and the third died of disease 7 years after radical lobectomy. The one patient who had negative mediastinal lymph nodes in the radical lobectomy specimen had a melanoma metastasis and will be described below.

In the period 3 to 5 years ago, 1 patient with a pulmonary metastasis had a radical lobectomy. This metastasis was from a renal carcinoma, and daughter metastases in the mediastinal lymph nodes were found. The patient died 6 months later of cerebral metastases.

Position of Tumor.—In the group of primary lung cancers, there were four in the right upper lobe, four in the right middle and lower lobe, and three in the left upper lobe.

In the group of metastases to the lung with postive extrapulmonic lymph nodes, there were three deposits in the right upper lobe, one in the left upper lobe, one in the right lower lobe, and one in the left lower lobe.

Multiple Primaries.—There were six double primary cancers, one of which was of the lung. Other sites included: 2 tongue, 2 breast, 1 larynx, and 1 lung.

Benign Conditions.—There were five radical lobectomies performed for tumor-like lung conditions that subsequently proved to be benign. These included 4 chronic pneumonias with atelectasis and/or abscess, and 1 lipoid pneumonia.

There were no complications or mortalities in this series.

COMMENT

In reviewing the results that have been listed in the previous section, it can be seen in Tables I and II that of 41 patients operated on either for primary

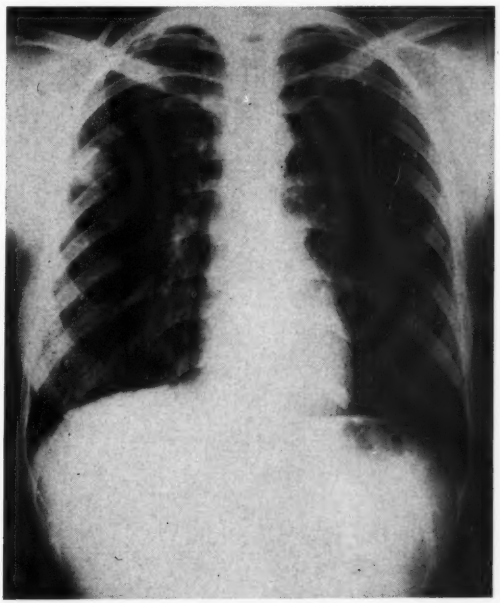

Fig. 8.—Roentgenogram showing metastatic teratocarcinoma of ovary in right upper lobe. Patient had right upper and middle lobe radical lobectomy. The specimen contained a daughter metastasis in the azygos vein node. Patient is without evidence of disease 8 years later.

or metastatic cancer to lung by means of a radical lobectomy, 17 (or about 42 per cent) had metastatic hilar and/or mediastinal lymph nodes when these were analyzed. In other words, 42 per cent had more cancer removed by a radical lobectomy than would have been removed had a simple lobectomy been performed.

Of these 17 patients with positive extrapulmonic lymph nodes, 8 were operated on 5 or more years ago, and 3 are alive without evidence of disease at this time. In these individuals, at any rate, it is quite probable that they owe their prolonged survival to the inclusion of these extrapulmonic lymph nodes, although there may have been contributory factors which are hard to analyze and document, such as host resistance. This term, host resistance, is an all-embracing one and may prove to possess several components. It attempts

to account for such phenomena as the rapid dissemination in 1 patient by an anaplastic or oat cell carcinoma of the lung, and the prolonged survival of another who harbors a microscopically identical tumor. It is probably influential in the survival of a patient who, in 1941, had a malignant melanoma excised from the skin of his back with an axillary dissection. Six years later after this last procedure, a right upper and middle lobe radical lobectomy removed a deposit bridging these two lobes. The mediastinal lymph nodes were negative. He is without evidence of disease 6 years and 4 months later.

Fig. 9.—Right radical pneumonectomy specimen with metal tags to label different node levels for identification by the pathologists.

In the past, the pathologic analysis of these specimens has been somewhat deficient in two respects: serial sectioning of all available lymph nodes was not done, although a single section for microscopic study was made through each one that was found; in addition, when these sections were studied under the microscope, no unusual efforts were made to hunt for or record vascular involvement by cancer unless there was invasion of relatively large veins. However, because the prognostic significance of lymph node and blood vessel involvement by cancer of the lung is now more firmly established, a more thorough study of these conditions is being made at this hospital. Included in this study is information about the levels and sites of lymph node involvement within the tracheobronchial tree and mediastinum (Fig. 9).

Just what extra chance for survival is afforded by the inclusion of the lymph nodes as part of a radical lobectomy or radical pneumonectomy is difficult to determine at present because of the limited number of procedures performed. The same is true in defining prognosis derived from the pathologic material alone.

Although a tentative attitude is necessary at this time, one fact seems to be evident: each case is a law unto itself, and survival probably is the result of the interplay of various pathologic states with the intangible forces of host resistance. It is therefore unwise to permit a single, isolated clinical or pathologic feature (for example, the threat of blood vessel invasion or the finding of a single mediastinal lymph node positive for cancer) so to dominate the picture that all aggression ceases. Instead, it would seem wiser to advocate an operation that attempts to eradicate most of the tangible elements in lung cancer, thus leaving less to chance. To our mind, a radical pneumonectomy and a radical lobectomy best fulfill this purpose.

SUMMARY

1. A radical lobectomy can be defined as an operation in which one or two lobes of an entire lung are excised in a block dissection with certain of their regional hilar and mediastinal lymphatics. This is to be distinguished from a simple lobectomy, which is an operation restricted to a lobe or lobes alone.

2. The pathologic rationale for including the hilar and mediastinal lymph nodes in either a radical lobectomy or a radical pneumonectomy is the finding based upon autopsy material that the lymph nodes are the most frequent sites of metastases from cancer of the lung. This fact is further confirmed by studies of lymph node specimens removed by these operations, at which time it was noted that metastases were present in them regardless of size, position, and histologic type of the primary tumor. In addition, autopsies on patients who had only a simple pneumonectomy or lobectomy for lung cancer revealed that there was recurrence in the ipsilateral mediastinal lymph nodes in 28 and 21 per cent, respectively.

3. The technique for removing each lobe with its regional lymphatics is described and diagrammed.

4. There are two major indications for a radical lobectomy. It is used to excise (a) a peripherally placed primary cancer of the lung for those who cannot tolerate a radical pneumonectomy, and (b) a lung tumor whose nature is ambiguous by reason of its association with a primary cancer at another site. It is also used for those tumefactions whose nature remains equivocal even at operation.

5. Results from 48 radical lobectomies, from 1951 to 1958, are listed. These were performed for 34 primary lung cancers, 9 metastases to the lung, and 5 benign, tumor-like conditions.

6. In those patients who had a radical lobectomy either for a primary cancer or a metastasis to the lung, 41.5 per cent had hilar and mediastinal lymph node metastases.

7. Eight patients were operated on 5 or more years ago, and 5 had positive mediastinal lymph nodes. Three of these are alive and well without evidence of disease.

8. The survival for 5 or more years of those who had excision of cancer-containing hilar and mediastinal lymph nodes is attributed in large part to the more extensive lymphatic dissection associated with a radical lobectomy.

9. It is believed that the careful study of excised hilar and mediastinal lymph nodes as part of a radical lobectomy or pneumonectomy provides a valuable basis for prognosis. The pathologic study of these lymph nodes, together with the knowledge of the histology of the primary cancer and evidence of blood vessel invasion by tumor are three essential aids in forecasting survival.

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PROGNOSTIC FACTORS AND RESULTS OF TREATMENT IN PYOGENIC PULMONARY ABSCESS

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THERE is little doubt that the advent of antibiotics has significantly affected the incidence and results of therapy of pulmonary abscess as well as other types of pulmonary infection. Acute bacterial pneumonitis is less common and its course is often aborted so that the secondary complications of empyema and abscess develop less frequently. However, certain patients fail to respond well to antibiotics and suffer a fatal outcome; others achieve resolution of the acute inflammatory process but are left with residual lesions necessitating operation to accomplish complete cure.

In order to examine more carefully this changing picture and to isolate, if possible, prognostic factors which would be useful in predicting the course of therapy, all patients with pulmonary abscess admitted to this hospital during the 10-year period prior to Jan. 1, 1959, were reviewed.

MATERIAL

During this period 90 cases of primary pyogenic pulmonary abscess were treated. Abscesses secondary to other conditions, such as carcinoma, pulmonary cysts, or tuberculosis, were excluded.

Most patients were from a low economic stratum or were indigents, and the proportion of elderly patients was high (Table I). Sixty per cent of the patients were over 50 years of age and over 35 per cent were over 60 years of age. There was a high incidence of significant associated disease which was of considerable import, both in leading to the development of abscess and in aggravating the course of the disease, frequently contributing to a fatal outcome.

Only a few patients were young and in good general health, factors which are reflected in the accumulated results.

The anatomic distribution of these lesions did not vary significantly from that in other reports, and the usual predominance in the right lung and in the upper portions of each lung was again noted. This distribution is most readily explained on the basis of aspiration although in only 9 cases (10 per cent) was there a history which clearly suggested aspiration.

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TABLE I. AGE DISTRIBUTION

YEARS	NUMBER
0- 9	2
10-19	0
20-29	6
30-39	10
40-49	18
50-59	22
60-69	19
70-79	11
80-89	2
Total	90

ETIOLOGY

Poor oral hygiene has been frequently incriminated as an etiologic factor. In this group of patients, 63 had definitely carious teeth, 16 patients had teeth in good repair, and 11 were edentulous.

Twenty cases followed an episode of acute bacterial pneumonitis, 5 being pneumococcal in origin.

In 9 cases there was a probable source of aspiration. Four patients had had preceding dental extractions, 2 had had tonsillectomies, 2 had had acute alcoholic bouts with stupor and emesis, and 1 patient had had a severe purulent sinusitis.

Four patients had had a preceding cerebrovascular accident with coma and 4 had chronic epilepsy, conditions which may well have predisposed to aspiration, although a definite history could not be elicited.

THERAPY

All patients had essentially similar initial medical therapy. In general, this consisted of appropriate antibiotics, postural drainage, and supportive measures. Bronchoscopy was performed in most cases, both diagnostically and therapeutically. Removal of bronchial plugs and associated forceful coughing following bronchoscopy frequently resulted in prompt evacuation of filled cavities, contributing to more rapid resolution of the acute inflammatory reaction.

X-ray studies were made at intervals and, as long as resolution of the inflammatory process progressed and the size of the cavity diminished, the medical program was maintained. Operation was considered whenever progress seemed to have become arrested or if there was residual cavitation after resolution of the acute inflammatory process. In 8 cases, the abscess was drained early in the patient's course, usually because of cavities which did not empty well or because of overwhelming toxic reaction.

RESULTS

Since all patients received similar initial therapy, the results of medical treatment can be divided conveniently into three categories based on the response to medical treatment. In the first group are the patients who achieved

a satisfactory result with medical therapy alone, with subsidence of inflammatory reaction and closure of cavities. In the second group are those patients who died while receiving medical therapy. In the third group are those in whom surgery was required to accomplish cure. The last group, of course, represents a large measure of medical success, since patients coming to resection have ordinarily obtained a subsidence of inflammatory reaction and operation is performed for control of residual lesions, particularly open cavities.

According to this classification (Table II), only 32 patients of 90 achieved a satisfactory result on medical therapy alone.

TABLE II. RESULTS

Patients treated medically:			
1. Satisfactory		32	(35%)
2. Fatal		21	(23%)
a. Due to abscess	11		
b. Due to associated disease	10		
Patients requiring operation		38	(42%)

Twenty-one patients died while undergoing medical therapy. Analysis of the causes of death in these cases revealed two important factors: the patient's age and concomitant disease. Table III illustrates the comparative age of patients who died and those who survived. There was a striking increase in mortality rate in elderly patients and in children; only 4 of 13 patients over 70 years of age and neither of the children (aged 2 and 4 years) survived.

TABLE III. RELATION OF AGE TO RESULT

AGE IN YEARS	MEDICALLY TREATED		SURGICALLY TREATED
	SATISFACTORY	FATAL	
0- 9	0	2	0
10-19	0	0	1
20-29	3	1	1
30-39	5	1	4
40-49	5	0	11
50-59	11	2	11
60-69	6	6	7
70-79	2	7	2
80+	0	2	0

In 11 of these 21 patients (12 per cent of the total number of 90), death was thought to be due primarily to the pulmonary abscess. Three patients died of direct complications, one death resulting from massive hemorrhage and two from acute perforation with bronchopleural fistula and empyema. Eight patients died as a result of uncontrolled infection; the ages of these patients were 76, 72, 80, 77, 88, 38, 63, and 57 years, emphasizing the effect of age.

In 10 patients death was thought due primarily to other causes: 4 patients died with cerebrovascular accidents, 2 patients had multiple systemic abscesses including cerebral abscesses which were thought to be the principal cause of death, 1 patient died in cardiac failure, 1 with enterocolitis, and 1 with massive gastrointestinal bleeding.

Thirty-eight patients required surgical treatment, 31 having resection of residual lesions and 7 having drainage of the abscess, usually in an attempt to control overwhelming infection. Four patients died of surgical complications following resection and 4 died of unremitting infection following drainage.

PROGNOSTIC FACTORS

Duration of Symptoms (Table IV).—The highest incidence of successful treatment with medical therapy alone was found in the group of patients who had symptoms less than 4 weeks prior to admission to the hospital. Over two thirds of the patients with symptoms present longer than 8 weeks prior to admission eventually required surgical treatment. No correlation could be established between the duration of symptoms and the likelihood of death.

TABLE IV. DURATION OF SYMPTOMS*

WEEKS	SURGICAL TREATMENT	MEDICAL TREATMENT
0- 4	7	26
4- 8	4	9
8-12	13	3
12+	8	6

*Only patients giving an accurate history are tabulated.

Bacteriologic Studies.—The distribution of organisms identified in the sputum of each group is shown in Table V. The most commonly identified organisms were *Staphylococcus aureus* and *Streptococcus viridans.* There was a scattered distribution among a number of types, each of which occurred in relatively small numbers. The most striking finding was in the group of staphylococcal infections, only 20 per cent of the patients obtained a good result by medical therapy alone, the remainder either requiring operation or succumbing to infection.

TABLE V. BACTERIOLOGIC ETIOLOGY*

	MEDICALLY TREATED		SURGICALLY TREATED
	SATISFACTORY	FATAL	
Staphylococcus aureus	6	9	15
Streptococcus viridans	18	6	17
Escherichia coli	5	6	3
Hemophilus influenzae	3	2	3
Pneumococcus	2	4	0
Anaerobic streptococcus	1	0	2
Enterococcus	2	0	1
Pyocyaneus	0	0	1

*In many cases more than one organism was present on repeated culture.

Location of Abscesses.—The location of abscesses in relation to prognosis is given in Table VI. Lower lobe cavities appeared to be appreciably more resistant to medical therapy. Less than 20 per cent of this group obtained a satisfactory result with medical therapy alone; approximately one half of these patients eventually required operation and an additional 30 per cent died while on medical therapy.

TABLE VI. LOCATION OF ABSCESSES

	MEDICALLY TREATED		
	SATISFACTORY	FATAL	SURGICALLY TREATED
Upper lobe	22	10	21
Middle-lingula	3	1	0
Lower lobe	7	10	16

Size of Cavity.—The relative size of cavities in these groups of patients is illustrated in Table VII. In a few cases original roentgenograms were not available, or for other reasons the size could not be estimated with reasonable accuracy. The most striking feature in this analysis was found in the results of treatment of large cavities. In patients with cavities 6 cm. in diameter or greater, only 2 of 20 achieved a satisfactory result by medical therapy alone; 9 patients succumbed to infection and 14 eventually required operation. The prospect for a successful result with medical therapy was much better in patients with cavities less than 2 cm. in diameter; nevertheless, even in these smaller lesions, a number of patients eventually required surgical treatment for complete obliteration of cavities.

TABLE VII. SIZE OF CAVITY

	MEDICALLY TREATED		
CENTIMETERS	SATISFACTORY	FATAL	SURGICALLY TREATED
0-2	11	7	7
2-4	11	3	12
4-6	8	2	4
6+	2	9	14

DISCUSSION

Despite the advent of antibiotic therapy and improvement of surgical techniques, pulmonary abscess continues to be a formidable disease. A rather wide variation in mortality rate has been noted in reported series,[1-4] in general tending to reflect the influence of the type of patient treated rather than the method of treatment.

It has been noted in this study that certain incidental factors are of serious import, particularly the age of the patient and associated or coincidental diseases. The percentage of good results of medical therapy in this series is lower than that reported by others,[1-5] probably reflecting the status of the patient and the abscess rather than a true variance in results, since the usual medical regimen is rather well standardized. It has become highly unusual for the patient who is reasonably young and in good general health to succumb to overwhelming infection associated with pulmonary abscess. In the present series, only 1 patient between the age of 10 and 50 died of pulmonary infection and 1 other patient in this age group died with a sudden massive hemorrhage. In approximately 50 per cent of the patients who died, death was thought due to other disease rather than to the abscess, which was being adequately controlled. Most of the patients whose death was thought primarily due to the abscess were elderly and debilitated patients. However, the 11 patients in

whom death was thought due to the abscess represent potentially curable cases. It is conceivable that early drainage of the abscess might have improved the results in this group.

Although Fox, Hughes, and Sutliff[2] have also noted an increased percentage of patients requiring operation with increased duration of symptoms prior to admission, relatively little attention has been given to other specific factors which are important in their influence on the course of the disease. In predicting the eventual outcome of the disease and anticipating which patients will be likely to require operation, the present analysis may be useful. If it can be predicted with a high degree of probability early in the course of the disease that operation will eventually be required, it is possible to prepare the patient physically and psychologically from the time of admission. Although none of the factors studied is an absolute prognostic guide, when taken in combination they provide a good index of the probable outcome and can lead to a more efficient therapeutic regimen.

SUMMARY

1. Ninety consecutive cases of primary pyogenic pulmonary abscess have been studied.

2. Thirty-two of 90 patients achieved a good result with medical therapy alone, 21 died while on medical therapy, and 38 patients required operation.

3. Of the 21 fatalities among patients on medical therapy, death was thought due primarily to the abscess in 11 cases which represent potentially curable patients with more effective treatment of the abscess. The other 10 patients succumbed to associated diseases.

4. Certain factors strongly indicated the likelihood of failure of cure by medical therapy alone. In descending order of certainty, these factors were as follows: (a) size of cavity 6 centimeters or more in diameter, (b) a history indicating onset of disease earlier than 8 weeks prior to admission, (c) staphylococcal infection, and (d) location of abscess in lower lobe of either lung.

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THE ANTERIORLY CRUSHED CHEST OR "STEERING-WHEEL INJURY"

An Improved Method for Sternal Traction

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CRUSH INJURIES of the chest are classified into (1) anterior, (2) lateral, and (3) posterior varieties,[1] and, of these, the anterior is particularly pernicious. Although it may occur in a variety of ways, the anteriorly crushed chest derives its pseudonym, "steering-wheel injury," from the fact that it occurs most commonly in automobile accidents when the driver of the vehicle is hurled forward by momentum over the steering post. It is the purpose of this report to present an improved method for applying sternal traction in this type of injury, to present a case in which the method was used, and to discuss briefly associated problems.

At the time of impact, sudden posterior displacement of the sternum produces fractures superiorly across the sternum, laterally across the ribs or costal cartilages on both sides, and occasionally inferiorly across the sternum as well, resulting in an unstable or "floating" section of anterior chest wall. Unless corrected, increasing respiratory insufficiency occurs, due to several associated mechanisms.

1. The "floating" segment of anterior chest wall moves paradoxically inward with inspiration and outward to neutral position with expiration, which produces a condition resembling a large sucking wound of the chest. Added respiratory effort is required in compensation.

2. Further reduction in ventilatory ability results from the attempt to minimize pain by minimizing excursions of the chest, with resultant inefficient rapid and shallow breathing.

3. Cough becomes inefficient because of the mechanical disruption of the thoracic cage, because of pain, and because of drugs given to alleviate the pain.

4. When cough is inefficient, secretions normally present are not efficiently expelled; but to make matters worse, reflex motor and secretory stimulation of the bronchial tree follows trauma to the chest, with resulting bronchorrhea, bronchospasm, and edema of the bronchial mucosa.[2, 3] Excessive production of pulmonary secretions coupled with marked interference with their evacuation results in "wet lung," followed by atelectasis and pneumonitis and progressive diminution in respiratory capacity and reserve.

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5. Ileus is an almost constant concomitant of severe chest injury. The gastric and intestinal dilatation produces elevation and restriction of the diaphragm and further loss of ventilatory capacity.

6. Mediastinal compression, immediate and acute, may be a significant cause of rapid deterioration. In addition, the inflow of blood to the heart through the great thin-walled veins of the chest, which is significantly assisted by the pumping and sucking action of fluctuations in the normally subatmospheric intrathoracic pressure, may be decreased to an alarming extent as negative pressure is reduced or, at times during the respiratory cycle, even abolished.[1]

The clinical picture is that of restlessness and anxiety, shock, cyanosis, tachycardia, rapid shallow breathing, and painful and ineffective cough. The lungs become filled with secretions. Movements of the floating segment may be slight at first, but, with gradually increasing embarrassment to respiration, marked increase in the amplitude of the paradoxical movement develops.

To reverse this disastrous sequence of events, stabilization of the floating segment must be accomplished. With stabilization, pain is minimized, ventilation improves, cough becomes effective, secretions are expelled, and the vicious cycle is interrupted and reversed. Methods of stabilization may be divided into (1) methods of internal fixation, (2) methods of external fixation by traction, and (3) Mörch and Avery's method of internal pneumatic stabilization by respirator.[18]

Internal fixation has been attempted by transfixion of the fractured ends of the sternum or ribs with Kirschner wires,[4] or by attempting to hold the fractured ends with wire or silk sutures.[5] Intramedullary nailing of the fractured ribs has also been attempted.[6] Methods of internal fixation have not been particularly satisfactory in cases of rib fractures, and even less so for the anteriorly crushed chest.[7] In such cases, internal stabilization is usually incomplete, inadequate, and insecure, and often lost as rib ends fragment or pins migrate.

Where feasible, traction is the most satisfactory way of restoring the integrity of the chest wall. As a mechanical result of the direction of the fracturing force and the direction of the resulting multiple fractures, the "floating" segment in anteriorly crushed chest is unstable inwardly; and traction applied in the opposite direction to that of the fracturing force will lock the fragments back in normal anatomic and functional position and produce a stable chest wall. The situation may be illustrated simply by comparing the crushed chest to a collapsible drinking cup, which loses its stability when collapsed, but becomes firmly rigid when the ends are distracted (Fig. 1). In addition to immediate correction of the fundamental disturbance, allowing more efficient ventilation, easier cough, and evacuation of secretions, the application of secure traction immediately expands the chest and increases the capacity of the lung for respiration and ventilation. Furthermore, traction allows healing in a more expanded chest position and makes for a better functional end result.

Various methods for the application of traction in anteriorly crushed chest injuries have been suggested. Towel clips and cervical tenacula have been used

to grasp the ribs or costochondral fragments adjacent to the sternum, and wires have been passed about the fragments.[8, 9, 10] Cervical tenacula have been applied across the sternum, with points in the opposite interspaces.[11, 12] Towel clips or cervical tenacula have been used to apply traction through drill holes in the anterior sternal table, with the points of the instruments approximated in the sternal marrow cavity. Specially designed tongs[13, 14] have been applied through drill holes in the anterior table of the sternum in similar manner. Wires have been threaded through holes in the anterior cortex. Various types of hooks have been screwed into the anterior cortex of the sternum to provide a purchase for traction, including coat-hanger hooks,[15] corkscrews and cup-hooks.[1] Hudson, McElvenny, and Head[7] have suggested an interesting method of soft-tissue traction using multiple subcutaneous Kirschner wires attached to spreader bows, adapted particularly to certain forms of the lateral types of crushed chest.

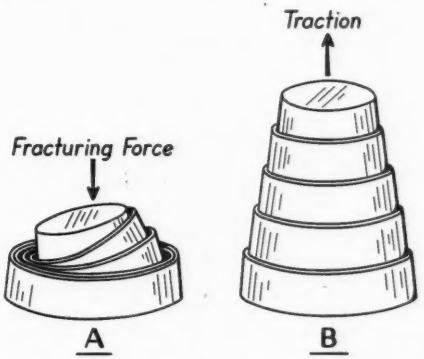

Fig. 1.—The anteriorly crushed chest may be likened to the collapsed drinking cup; with traction the fragments lock and stability is achieved.

It is apparent, from the very variety of suggested methods of sternal traction, that none is completely satisfactory and universally adaptable. One of the major faults with the numerous devices has been the lack of security. Fragmentation of bone and loss of anchorage often occurs with the application of prolonged and adequate traction force and this leads to the necessity for reducing and compromising with the weight applied. Leverage may be considerable and may cause fragmentation and loss of purchase in spite of meticulous and delicate care.

PROCEDURE

We have simply and effectively resolved these mechanical difficulties in applying and anchoring traction devices by passing a Kirschner wire *underneath* the sternum in the center of the floating segment, through the retrosternal space,[16] using the wire as a yoke through which traction is applied. This method provides strong, secure, and dependable anchorage through the full thickness of the sternum rather than through one fragile cortex, permitting application

of as much force as desired without fear of distraction or loss of purchase as the days progress. There is no leverage to loosen the purchase, and the direction of pull can be changed as desired along the axis of the Kirschner wire, affording easy mobility to the patient in bed, who can sit up or lie down while traction is continuously exerted. The procedure is as follows:

A generous incision is made transversely across the chest over the center of the floating segment and carried to the intercostal spaces on either side. The internal mammary vessels may be ligated, or may be avoided by staying close to the sternum. A grooved director is bent about 1.5 cm. from its tip to a 60-degree angle, and the shaft of the director itself is gently curved concavely, as illustrated in Fig. 2.

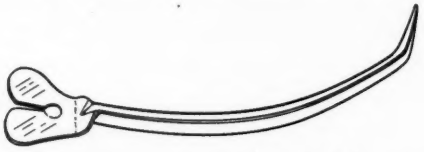

Fig. 2.—A grooved director is bent as illustrated.

The director is then passed under the sternum in the center of the floating segment, with care to avoid entering either pleural space. The tip of the director is maintained in apposition to the body of the sternum at all times. A flexible Kirschner wire is passed through the groove in the director, and the director is then removed, leaving the wire in place (Fig. 3). The ends of the wire are carefully secured with a Kirschner wire spreader bow, and traction immediately applied. The patient's condition improves at once. The incision is closed, and weights applied through a system of pulleys. The patient is allowed considerable mobility in bed. There is no danger of leverage loosening the purchase, nor of excessive weight tearing out the moorings. We have used 20 pounds of traction initially, and continued traction for about 3 weeks with gradual reduction in weight terminally. The procedure may be performed under local anesthesia, although we have preferred the complete control of respiration afforded by endotracheal intubation under topical anesthesia, followed by light general anesthesia, with suctioning of the tracheobronchial tree and administration of 100 per cent oxygen.

Patients in whom the chest wall is so comminuted that traction will not lock the fragments into position can now be successfully treated by "internal pneumatic stabilization," using the brilliantly conceived Mörch respirator attached to a tracheotomy.[18] This method represents a singularly striking advance in the therapy of severe, previously fatal, crushing injuries. While truly lifesaving in these cases, the method is associated with morbidity and complications, and requires constant careful and meticulous attention to detail over a prolonged period of time. In situations where adequate stabilization can be achieved by traction, this is much to be preferred, being simple and easier, and returning the patient more rapidly and completely to a self-sufficient physiologic state.

Next in importance to actual stabilization of the chest is tracheotomy, and this should be routinely employed. Its value has been amply demonstrated.[17]

Tracheotomy allows aspiration of secretions, diminishes dead space, and diminishes the effort of breathing by removing nasal and pharyngeal resistance. Tracheotomy should complement, not replace, correction of the basic defect by skeletal traction, which remains of prime importance and the cornerstone of therapy. Nasogastric or intestinal intubation to relieve gastric dilatation and abdominal distention secondary to the paralytic ileus is an important measure.

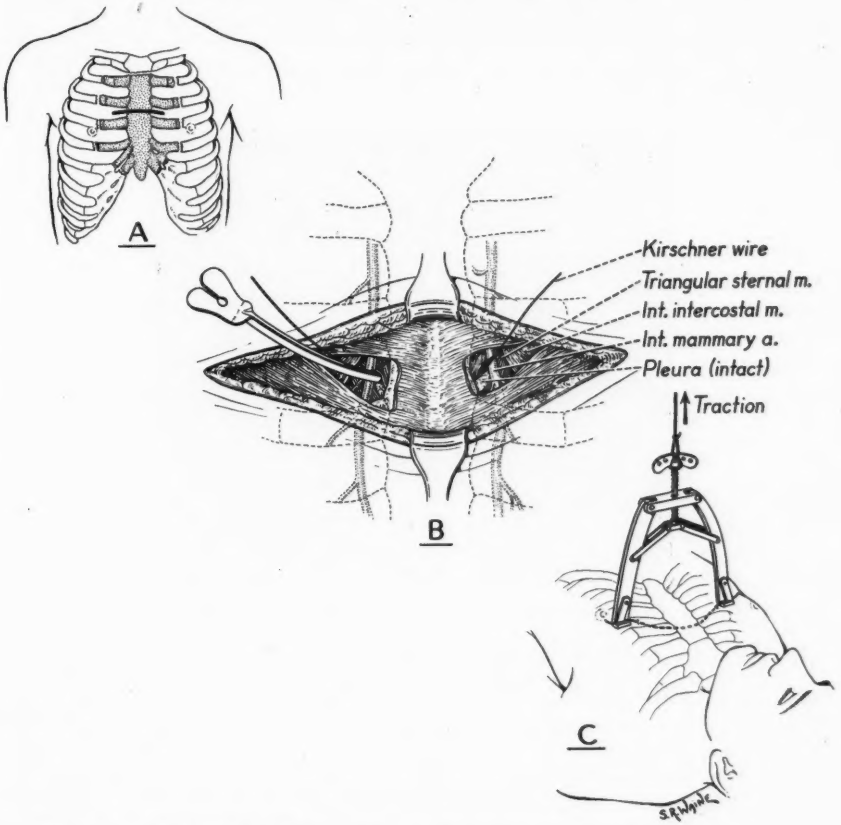

Fig. 3.—*A*, The skin incision in relation to the fractured segment.

B, The grooved director has been passed from one side of the sternum to the other, and the Kirschner wire has been passed through the groove. Following this, the director is carefully removed leaving the wire in place.

C, A Kirschner wire spreader is securely attached to the ends of the wire and continous traction immediately applied.

Antibiotics are indicated to prevent and treat pneumonitis. In addition, supportive therapy must be given as necessary, including blood, intravenous fluids, oxygen, and careful tracheotomy care. Other serious injuries may be associated with the crushed chest, including pneumothorax, hemothorax, tracheal or bronchial rupture, cardiac laceration and tamponade, diaphragmatic rupture, and extrathoracic injuries and must be treated as indicated.

CASE REPORT

The following illustrative case is presented:

Mr. W. K., a 56-year-old white man, was admitted to the Jewish Hospital of Cincinnati on July 24, 1956, following an automobile accident on the preceding day in which he had sustained an anterior chest injury and fracture of the left tibia. He had been observed elsewhere and had seemed at first to be quite comfortable. In the ensuing hours, however, he had developed increasing difficulty breathing, and when finally transferred to this hospital was in extremis from respiratory insufficiency. Examination revealed marked cyanosis and dyspnea, and striking paradoxical motion of the anterior chest. The lungs were filled with secretions. X-ray studies revealed marked gastric dilatation, and a haziness in the right lower chest attributed to fluid.

Fig. 4.—Photograph of the apparatus in use.

The patient was taken immediately to surgery with a diagnosis of anteriorly crushed chest. A Levine tube was passed and the gastric dilatation relieved. An endotracheal tube was passed and the patient was lightly anesthetized with Pentothal sodium. One hundred per cent oxygen was administered, and copious bronchial secretions were aspirated. With the patient under respiratory control, following sterile preparation and draping, a flexible Kirschner wire was passed beneath the sternum according to the method just described. The incision was closed, and the ends of the wire were attached to a Kirschner wire spreader (Fig. 4). Respiration improved immediately with the application of traction. A complementary tracheotomy was then performed. The patient returned to his bed markedly improved.

Convalescence was uneventful. The severe wet lung present on admission cleared rapidly. The patient was treated with intravenous fluids and antibiotics, and was maintained on nasogastric suction for 2 days. Twenty pounds of traction were applied at first, and gradually reduced during the progress of the convalescence. The Kirschner wire was removed on Aug. 14, 1956, 21 days after operation, with the chest remaining completely stable. An interlobar hematoma of considerable size was present on the right side and was treated conservatively, with subsequent complete resolution. The tibial fracture healed satisfactorily following closed reduction. The patient has remained well following discharge from the hospital.

SUMMARY

Crush injuries of the chest are a particularly devastating type of injury, and lead rapidly to respiratory insufficiency because of (1) paradoxical respiration, (2) shallow rapid breathing, (3) inefficient cough, (4) accumulation of secretions, (5) paralytic ileus, and (6) cardiovascular embarrassment. These mechanisms are directly related to the loss of integrity of the thoracic cage. Immediate restoration of that integrity is essential. This is usually best accomplished by skeletal traction. An improved technique for skeletal traction in anterior crushed chest injuries is presented, involving the passage of a Kirschner wire beneath the sternum, the attachment of a Kirschner wire spreader bow to the ends of the wire, and the application of traction through the entire thickness of the sternum through the yoke so formed.

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INTRAMURAL ABSCESSES OF THE ESOPHAGUS: A COMPLICATION OF FOREIGN BODIES

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ENDOSCOPISTS have reported drainage of acute localized phlegmons of the esophagus. Apparently these cases are rare since relatively few have been recorded. Experience with an intramural abscess of the esophagus, which clinically simulated a neoplasm and was treated by subtotal esophagectomy, aroused interest in the subject.

A review of the literature disclosed that previously reported parietal abscesses of the esophagus involved the presence of foreign bodies. This significant relationship is emphasized by the following report.

CASE REPORT

Miss K. O., a 56-year-old secretary, was admitted to the hospital on Nov. 25, 1957, with the complaint of retrosternal pain of 24 hours' duration. The previous day a constant, nonradiating retrosternal pain developed after the patient, who seldom ate meat, had a portion of a pork chop. The pain was markedly accentuated by swallowing but not by exertion or deep breathing. It was unrelieved by coughing or change of position. Patient stated that after the onset of the pain she had choking sensations which continued until the forenoon of the day of admission. At that time a large amount of thick, brown-colored, fluid-like material appeared in her throat. It did not result from a cough. Patient was relieved but was weak after this incident. She also reported some choking sensations which had occurred 4 to 5 months previously. There were no other symptoms related to the thoracic organs. There had been no loss of weight; her usual weight varied between 36.3 and 40.9 Kg.

There had been no previous serious illnesses or operations. She had been treated earlier in the year for an "infected nose" by an otolaryngologist. In January, 1953, patient had been hospitalized for swelling of the face and ankles due to secondary anemia and malnutrition. In August, 1956, she had been rehospitalized for a dull, intermittent pain in the left upper quadrant of the abdomen and left lower chest. Laboratory and roentgenologic studies resulted in the diagnoses of atropic gastritis and mild malnutrition. After fluoroscopy with a barium meal, the esophagus was reported normal. A history suggestive of sinusitis was elicited.

At physical examination the temperature was 36.8° C.; pulse, 78; respiration, 18; blood pressure 140/70 mm. Hg. The nasal and pharyngeal mucosae were injected and an apical systolic murmur was heard. The blood counts, urinalysis, and serological and blood chemistry determinations were essentially normal. The albumin/globulin ratio was 1.5:1. The corrected sedimentation rate was 30 mm. An electrocardiogram was normal. A thoracic roentgenogram made on November 26 revealed bilateral apical pleural thickening of a moderate degree and a few fibrotic strands in the left infraclavicular area. The heart was increased in size

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(17 per cent by measurement). There were mild sclerotic and degenerative changes in the thoracic aorta. A liquid barium esophagogram on the same day demonstrated almost complete obstruction at the junction of the middle and distal thirds of the esophagus; a very small amount of barium escaped past a filling defect which measured 5 cm. in length and

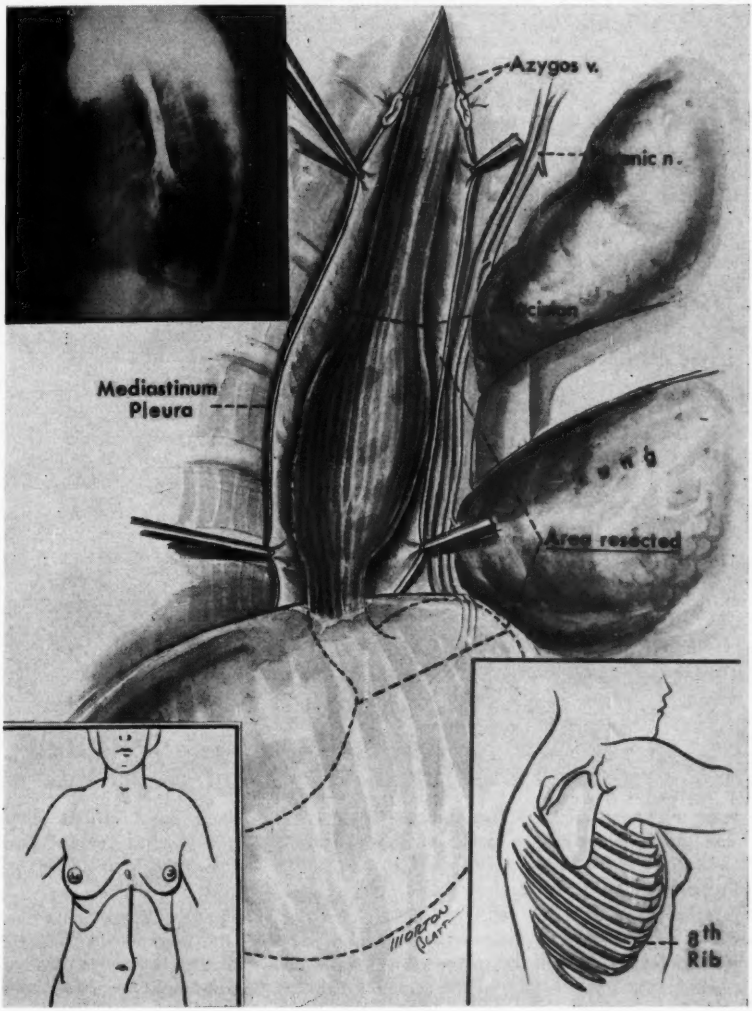


Fig. 1.—Artist's diagrammatic representation of lesion at the time of surgery, with preoperative roentgenogram.

appeared to be within the lumen. A slight dilatation of the esophagus was present, immediately proximal to the filling defect (Fig. 1).

Course in the Hospital.—On November 27 at esophagoscopy a foreign body (gray-colored bolus of food) was seen and removed from the middle of the esophagus. Distal to this site the normal-appearing mucosa bulged into the lumen, especially from the right side.

The lumen was thereby so narrowed that the esophagoscope was not advanced further. A biopsy showed normal esophageal mucosa. The foreign body was reported to be partially digested material of plant and animal origin; the latter included a small fragment of bone. The esophagogram was repeated on November 30, at which time an obstruction, almost complete, was again seen at the level of the eighth thoracic vertebra. An intraluminal-type defect was noted, with very little barium passing beyond the area. The roentgenologist concluded that a neoplasm of the esophagus was present. Intravenous fluids were given daily preoperatively.

On December 2, a right thoracotomy revealed an 8 cm. cylindrical, smooth, firm mass in the esophagus below the arch of the aorta (Fig. 1). Its pink posterior surface was flecked with yellow. The mass was continuous above and below with normal-appearing esophagus. After the azygos vein was ligated and divided, the mass and distal esophagus was freed with ligation of vessels from the aorta and hilar areas. A small amount of granulation tissue was present in the posterior mediastinum adjoining the esophageal mass. The patient was then moved to a semi-supine position, and the peritoneal cavity was opened by a left paramedian incision. The stomach was mobilized by separation of the gastrohepatic and gastrocolic ligaments, with division of the vasa brevia artery, the left gastric artery, and the left gastro epiploic artery at its origin. The coronary ligament of the left lobe of the liver was divided, the esophagus freed from the hiatus, and the right crus of the diaphragm divided. A Heineke-Mikulicz pyloroplasty was then performed. The esophageal hiatus was enlarged to permit the introduction of three fingers. The liberated stomach was then pushed and simultaneously pulled up into the thorax through the enlarged esophageal hiatus. The abdominal incision was then closed. The proximal portion of the stomach was transected and the distal end closed. The esophagus was transected proximal to the tumor mass. The latter, plus the distal esophagus and gastric cardia, were removed as one specimen. An esophagogastrostomy was then performed at the level of the sixth thoracic vertebra, and the stomach was sutured to the mediastinum and esophageal hiatus. Two intercostal drainage tubes were placed in the right pleural space, and the thoracic incision was closed in layers. The patient's status throughout the procedure was exceptionally good; she left the operating room in satisfactory condition.

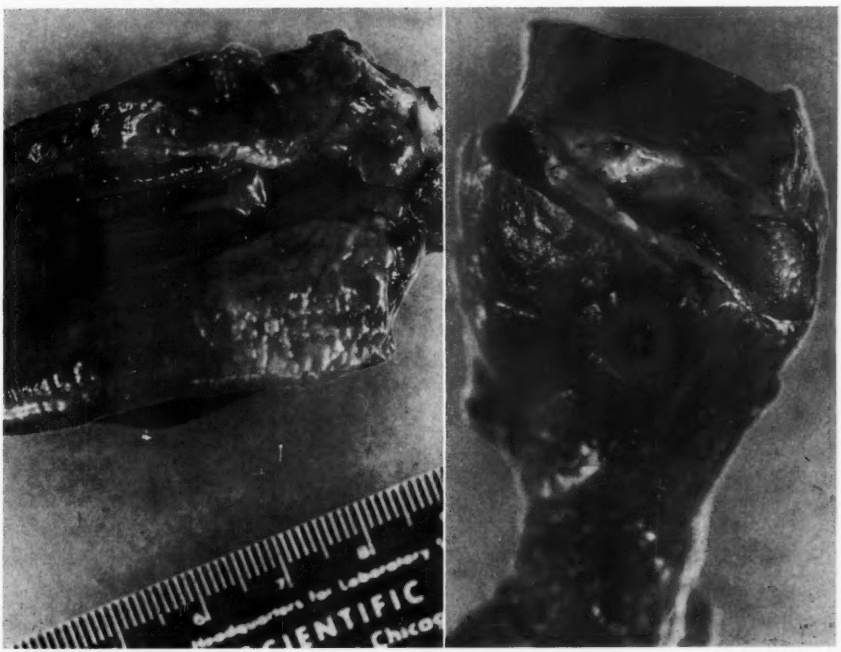
The surgical specimen was submitted to the Pathology Department without any attempt to incise or explore it. The pathologic report can be summarized as follows: The specimen consisted of the distal portion of the esophagus and the attached cuff of the fundus of the stomach. After sectioning, an irregularly shaped superficial ulcer, 2.0 by 0.5 cm., was seen in the mucosa, with its longer dimension along the axis of the esophagus. This was located 1 cm. below the superior edge of the resection and extended only through the mucosa and submucosa. The otherwise intact mucosa had a bluish-gray, smooth, glistening surface. Within the wall of the esophagus was a firm mass, measuring 7.0 by 3.0 by 1.2 cm. Cut section through this mass (Fig. 2) revealed smooth, glistening gray and tan surfaces, in the center of which was a small cavity (0.4 cm. in diameter) filled with purulent material. The ulcer was found to be connected to the abscess by a pin-point sinus tract.

Microscopic sections revealed an ulcer extending through the mucosa into the submucosa. The base of the ulcer was covered by many polymorphonuclear neutrophils. Beneath was a narrow tract of necrotic tissue, heavily infiltrated by polymorphonuclear neutrophils. This, in turn, was continuous with an abscess cavity containing many polymorphonuclear neutrophils and necrotic tissue. There was a suggestion of foreign material. At the margins of the ulcer and of the abscess, there was actively proliferating granulation tissue infiltrated by moderate numbers of lymphocytes and polymorphonuclear neutrophils. This tissue formed the outer margin of the specimen. The smooth muscle of the muscularis showed marked degeneration in the zone surrounding the abscess. A few foreign body giant cells were noted in the submucosa adjacent to the ulcer. There was a diffuse infiltration by polymorphonuclear neutrophils and lymphocytes of the

muscularis about the lesion. In one section a space lined by a thin layer of condensed hyaline-like material was continuous with the abscess cavity. The surrounding tissue in some areas was infiltrated with small foci of polymorphonuclear neutrophils.

Diagnosis.—(1) Intramural abscess of the esophagus; (2) ulcer of esophagus, superficial, subacute.

The Chief Pathologist was consulted about the report of the specimen. He stated that the abscess was essentially an acute process and not over 2 weeks old. Inasmuch as the granulomatous reaction was minimal it could not be called a granuloma. There was no evidence to suggest that the ulcer was of the peptic type or that a diverticulum had been present in the area of the lesion.



A. *B.*

Fig. 2.—*A*, Specimen opened, demonstrating thickened esophageal wall and small ulcer with some mucopurulent material present in it. *B*, Wall of specimen sectioned showing the thick wall of the abscess.

Postoperatively, the patient's recovery was slow, but progressive. Some pneumonitis developed in the left upper lobe and then cleared. A roentgenologic study made on December 23 demonstrated retention of barium in the thoracic stomach after 3 hours. On January 30, only a small residual of barium remained in the stomach when the test was repeated.

It was necessary to treat a painful calcified bursitis of the right wrist as well as a painful left hip before the patient was discharged from the hospital on February 15.

Dr. F. C. Murphy continued to see the patient after she left the hospital. On Nov. 12, 1958, she reported to the writer that there was an occasional pain in the mid-epigastrium an hour or more after eating soft regular food. Her weight had been 31.4 Kg. in the summer, but at that time was 36.3 Kg. (height,

TABLE I. CASES OF INTRAMURAL ABSCESS OF THE ESOPHAGUS

CASE NO.	AGE	SEX	DURATION	ETIOLOGY	LEVEL	CLINICAL COURSE	AUTHOR
1	62	F	4 da.	Chicken bone	C_7 vert.	F.B.* removed; abscess drained; satisfactory x-ray in 9 days	Vistreich[13]
2	49	M	3 da.	Fish bone	18 cm.	F.B. removed; abscess drained; well in 3 days	Kramer[7]
3	49	M	10 da.	Fish bone	16 cm.	F.B. not seen; abscess drained; well in 2 days	Kramer[7]
4	?	?	8 wk.	Beef bone	?	Abscess drained; F.B. removed later; healed satisfactorily	Barker[3]
5	3	?	6 wk.	Coin (six pence)	12 cm.	F.B. removed; abscess drained; satisfactory x-ray in 3 weeks	Barker[2]
6	70	M	3 da.	Pork steak bone	C_6 vert.	F.B. removed after rupture of abscess in a diabetic patient	Schwartz[11]
7	26	M	4 da.	Denture	C_6 vert.	F.B. removed; abscess resolved	Matheson[8]
8	67	F	4 da.	Fish bone	21 cm.	Abscess drained; no F.B. seen; well in 4 days	Handke and Bade[6]
9	45	F	3 da.	Fish bone	19-20 cm.	Abscess drained; no F.B. seen; mediastinotomy later	Alpin[1]
10	45	F	4 da.	Lamb bone	15-16 cm.	Abscess drained; F.B. passed; well in 4 days	Alpin[1]
11	8	M	8 da.	Calf bone	12-14 cm.	Abscess drained; no F.B. found; well in 8 days	Alpin[1]
12	24	M	5 da.	F.B. with food	30 cm.	Abscess drained; stricture resulted, bougienage required	Clark[4]
13	?	M	3 da.	Bone	C_7 vert.	F.B. removed; later abscess drained	Robinson[10]

*Foreign body.

164.5 cm.). She stated that she had returned to her full-time duties as secretary on April 21. On May 6, 1959, the patient reported that she weighed 38 Kg. and had not been absent from work in over a year.

DISCUSSION

Only a few cases of acute localized phlegmons of the esophagus have been reported. According to Graham, Singer, and Ballon,[5] von Hacker was the first to cure a patient with a circumscribed phlegmon of the esophagus by esophagoscopically removing the bone which caused the lesion. Handke and Bade[6] cited Seiffert as the first to describe the incision of an esophageal abscess via the esophagoscope. Graham and his associates reported Guisez as curing 5 patients who had almost complete stenosis of the esophagus because of marked bulging of the esophageal wall due to abscess formations. Kramer[7] stated that Guisez treated 8 patients with one death (patient was first seen when in extremis). Apparently no ill effects or stenosis occurred. Albrecht and Hertzog, also cited by Graham, obtained good results using the same techniques. One of 3 patients, treated similarly by Gottstein, died of mediastinitis on the eighth day.

The literature revealed, in addition to those already mentioned, 13 cases of acute esophageal abscesses (Table I). These occurred in patients from 3 years of age to 67, with nearly an equal number of males and females. It is significant that the abscesses developed, and were large enough to cause obstruction, in a number of the cases in less than 5 days. The etiological agent in all cases was the incomplete swallowing of a foreign body, usually fish or meat bones, but a coin (six pence) and a denture were also causative factors. The abscesses were located at varying levels in the esophagus, but were mainly between 12 and 30 cm. from the upper incisor teeth. In some cases the foreign body was not found. When seen, it was removed. Drainage of the abscesses was usually accomplished with the aid of an esophagoscope. Stenosis developed in 1 case (at 30 cm.) and bougienage was required subsequently. In 2 of the cases, the periesophageal tissues were probably involved, but an attempt has been made to cite only localized phlegmons. A collar mediastinotomy was performed in a complicated case reported by Alpin.[1]

Terracol and Sweet[12] have in their monograph a brief, well-written chapter including abscesses of the esophagus. In the bibliography, a case of traumatic abscess reported by Nys[9] is given.

In the differential diagnosis of obstructive lesions of the esophagus, a parietal abscess should be considered, especially when a foreign body has been present. As to definitive treatment, drainage, utilizing an esophagoscope, is a more conservative type of surgical procedure. However, subsequent bougienage may be required for cicatricial stenosis of the esophagus. In advanced lesions, the wall of the esophagus might be destroyed to the extent that resection is indicated.

The possibility of an intramural or parietal abscess of the esophagus was not considered preoperatively, or during surgery, in the case just described. The patient was afebrile and had a normal total and differential leukocyte

count on admission. The esophagogram suggested a neoplasm, as was true in one of Kramer's cases (Case No. 3, Table I). Undoubtedly a stricture would have resulted if a drainage procedure had been done at surgery. Thus, resection prevented subsequent need for bougienage. This patient probably would not have tolerated such procedures repeatedly. A drainage operation at the time of thoracotomy is not without other complications than stricture. Mediastinal suppuration, with its ramifications, is one of several possibilities. It is fortunate that the abscess did not perforate into the mediastinum to any extent.

The mucosa of the esophagus is apparently easily injured by foreign bodies. Bacteria then have a portal of entry to the underlying tissues, which have very little resistance to contamination, and infection and suppuration develop rapidly.

The present case also illustrates the need for a correct histologic diagnosis before refusing, or failing to recommend, surgery in patients with obstructive lesions of the esophagus. Roentgenologic examinations are helpful but may be misleading.

SUMMARY

An intramural abscess of the esophagus has been described. The benign condition simulated a neoplasm roentgenologically before surgery and was treated successfully by resection. The reported cases of acute localized phlegmons of the esophagus have been summarized and their association with foreign bodies has been emphasized.

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A CASE OF PURE ENDOBRONCHIAL CHONDROMA

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THE PURPOSE of this paper is to report a case of a pure chondroma of endobronchial origin, occurring in a middle-aged woman. These tumors arising from this site are extremely rare and only a few cases have been reported in the literature.

Almost all these cartilaginous tumors have been called "hamartomas," a term which originated from Albrecht,[1] in 1904, who stated that "hamartomas are tumor-like malformations in which occur only an abnormal mixing of the normal components of the organ. The abnormality may take the form of a change in quantity, arrangement and degree of differentiation, or may comprise all three. The deduction to be drawn from histologic examination of these formations is that they have originated in an abnormal mixing of the elements or from disturbances of their development." The term as originally coined by Albrecht was applied to such tumors of the liver.

CASE REPORT

A 55-year-old white woman was admitted to St. John's Riverside Hospital on May 12, 1958, with a chief complaint of fever and cough. The patient had been in good health until approximately 10 days prior to admission, when she noted the onset of fever accompanied by muscular aching and cough. The fever was intermittent with daily temperature rises. The cough was productive of a moderate amount of tan-colored sputum which occasionally was blood-streaked. The patient was treated at home by a physician with supportive therapy and, when she failed to improve, hospitalization was advised.

A review of her past history revealed that she had had several similar episodes within the previous 6 months. The patient had no wheezing in the chest but had mild dyspnea on exertion. She complained of pain in the right upper chest region anteriorly and laterally which was pleuritic in nature. Her systemic review was noncontributory to the present illness.

The patient had had pleurisy in the right chest approximately 10 years prior to this admission. A tonsillectomy was performed at the age of 18 and she had the usual childhood diseases. She was a relatively heavy smoker, admitting to more than one pack per day. She had no history of allergy. Positive physical findings were limited to the chest where there was evidence of decreased breath sounds in the right upper lung field anteriorly and posteriorly with dullness and a positive cough sign.

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Initial treatment consisted of erythromycin, expectorants, aspirin, and sedation. An electrocardiogram was within normal limits. A chest roentgenogram (Fig. 1) revealed flocculant mottled infiltrate which involved the posterior segment of the right upper lobe. A temperature of 103° F. occurred on the first day, with return to normal in 5 days. The cough, however, persisted, as did the pain in the chest. Six days following admission repeat x-ray studies (Fig. 2) showed further atelectatic changes in the right upper lobe region and neoplasm was suspected.

Bronchoscopy was performed on January 24 under local anesthesia and a mass was found which occluded the right upper lobe orifice completely. The mass was grayish-pink and appeared to be fungating. A biopsy showed bronchial mucosa with hyaline cartilage beneath the mucosa. It was thought that the biopsy had been inadequate.

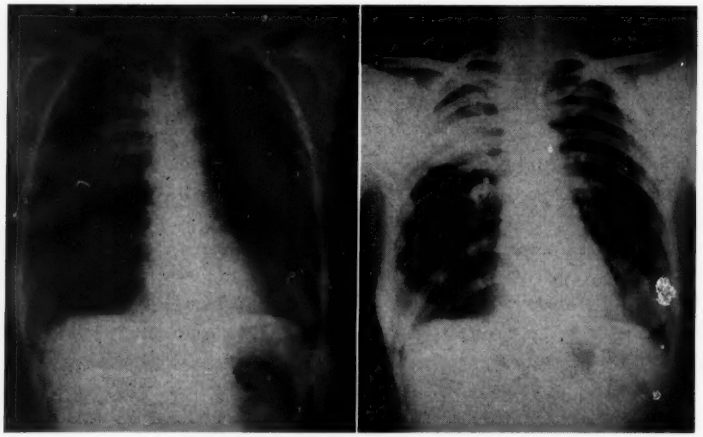

Fig. 1. Fig. 2.

Fig. 1.—Roentgenogram taken on admission discloses a flocculant mottled infiltrate involving a segment of right upper lobe.

Fig. 2.—Roentgenogram made 6 days later discloses atelectatic changes in the right upper lobe region with the possibility of neoplasm as suggested by the roentgenologist.

Because of the clinical diagnosis of bronchogenic carcinoma, an exploratory thoractomy was performed on May 27, 1958. Due to the absence of an absolute pathologic diagnosis, the main right upper lobe bronchus was opened posteriorly and the mass was again noted. Several punch biopsies were taken and frozen section examination again revealed only cartilage. The diagnosis of benign chondroma was made and the incision in the bronchus was extended into the apical segmental bronchus where it was found that the mass was a chondroma on a very small pedicle originating in the right upper lobe apical bronchial segment. The pedicle was excised with an ovoid incision through the mucosa. The area from which the tumor had arisen was curetted with a small curette. The bronchus was closed with interrupted 4-0 silk sutures and a pedicle graft of pleura was placed over the incision in the bronchus.

The patient made a completely uneventful convalescence, with complete expansion of the right upper lobe. She was discharged on the ninth postoperative day in good condition.

The pathologic report was as follows: the gross specimen consisted of an irregular, small, whitish, firm chondroma, glistening upon sectioning and measuring 0.8 by 0.6 by 0.6 cm.

Microsections showed a typical chondroma partially lined by the normal mucosa of the bronchus. The cartilage was composed of a uniform transluscent matrix with evenly situated oval lacunae containing small cartilaginous cells which were adult in type. There was a complete absence of other types of tissues which might warrant classification of this tumor as a hamartoma. Malignancy was absent.

DISCUSSION

According to McDonald, Harrington, and Clagett,[2] it cannot be denied that pure chondromas of the lung occur, although they have had no experience with them as such. These authors indicate that cartilaginous tumors in the lungs are occasionally referred to as chondromas, when the other elements, such as epithelium, fat, and smooth muscle, are incorporated in the tumor. These are not true chondromas.

According to Rubin and Beckman,[3] location of a chondromatous hamartoma in a bronchus is uncommon. They reported the fourteenth case of this type and they were unable to find a single pure chondroma in 8,800 necropsies reviewed. Because of the close relationship of pure chondromas of the bronchus and hamartomas, these tumors, for convenience and symptomatology as well as prognosis, should be considered as similar tumors.

In an article by Nissen and his associates,[4] cartilaginous tumors of the lungs are frequently encountered at autopsy. They are usually peripherally located, are of hamartomatous nature, and are not clinically significant. Cartilaginous tumors arising from the large bronchi are of clinical importance because they give rise to signs and symptoms which may be easily interpreted as due to carcinoma but are amenable to curative surgical procedures. These authors found only 20 cases in reviewing the medical literature.

According to Young and his associates,[5] endobronchial hamartomas have occasionally been removed through the bronchoscope and none has been known to recur. They often produce recurrent pneumonitis and atelectasis and make it desirable to remove the involved segment. The most frequent symptoms have been cough and hemoptysis. The roentgenograms in the cases of Young were not diagnostic of bronchial tumors. A review of the literature by these same authors disclosed 24 cases of endobronchial hamartomas. Of the 24 cases reported, 13 arose in the right lung and 11 in the left lung. Seven of the tumors were incidental findings at autopsy and the other 17 produced symptoms. There has been no instance of a bronchial chondroma becoming malignant.

In a 1958 publication, Hodges[6] found that in 200 reported cases of hamartomas of the lungs, the tumor was endobronchial in 33 and in the lung parenchyma in the remainder.

SUMMARY

This is the report of a middle-aged white woman who was admitted to the hospital mainly for cough and fever. The symptoms and signs, including x-ray interpretations, were those of a neoplasm, probably carcinoma. However, upon opening the bronchus at thoracotomy, a pedunculated firm tumor was found

completely obstructing the lumen of the right upper lobe. Frozen and paraffin sections disclosed it to be a pure chondroma. On the ninth day following removal of the growth, there was complete expansion of the right upper lobe and the patient was becoming symptom free.

This case seemed to follow the pattern of most of the cartilaginous endobronchial tumors reported in the literature, with obstructive bronchial signs followed by atelectasis, and complete cure following the removal of the tumor.

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ACQUIRED TRACHEOCELE: AN UNUSUAL LESION

Surgical Resection in a Case Associated With Trachiectasis and Bronchiectasis

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Among the benign conditions affecting the tracheobronchial tree, diverticulum, or tracheocele, represents one of the more uncommon. Essentially, this lesion consists of an out-pouching, or sac, formed by membranous trachea at its junction with the cartilagenous rings (the cornu). The more common congenital variety is usually small and narrow-mouthed, probably representing a supernumerary bud which has failed to become isolated as a bronchogenic cyst. Typically, these occur about 4 to 5 cm. below the true vocal cords, most often present at the right cornu, are clinically silent, and sometimes multiple.

The acquired variety, on the other hand, is usually wide-mouthed, tends to be larger in size, may occur at any level, and often produces esophageal compression. Chronic infection in glands of the posterior tracheal wall with progressive destruction of elastic tissue has been postulated as its etiology. Trachiectasis and/or bronchiectasis may be associated and secondary to it. Indeed, the signifiance of such a diverticulum resides not only in its ability to produce cough, but more importantly in its acting as a reservior for secretions which may result in chronic reinfection of the tracheobronchial tree.

Although described by Rokitansky[1] over 100 years ago, relatively few cases of tracheocele have been reported. The great majority of these has been of the congenital type, incidentally noted at necropsy or bronchographic examination. Drymalski,[2] Mackinnon,[3] Goldman,[4] and others[5, 6] have reviewed the various aspects of this lesion and presented cases. In 1944, Addington and coworkers[7] reported an acquired tracheal diverticulum in a 32-year-old man who had complained of cough, dyspnea, and wheeze for 8 years. This was a wide-mouthed sac demonstrated at bronchoscopic examination, produced esophageal compression, and was accompanied by basilar bronchiectasis.

Three surgical cases have been recorded in which tracheocelectomy was performed. In 1943, Christmann and Albano[8] described cervical excision of a plum-sized sac communicating between the second and third tracheal rings. During the previous 15 years it had been intermittently palpable in the right

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supraclavicular fossa and had become the site of recurrent infection with purulent sputum. In 1950, Mathey and Lemoine[9] operated upon a 16-year-old girl who was found to have a congenital tracheoesophageal fistula (without atresia) as well as a congenital tracheocele. The former entered the trachea 3 cm. above the main carina and was divided and closed. The

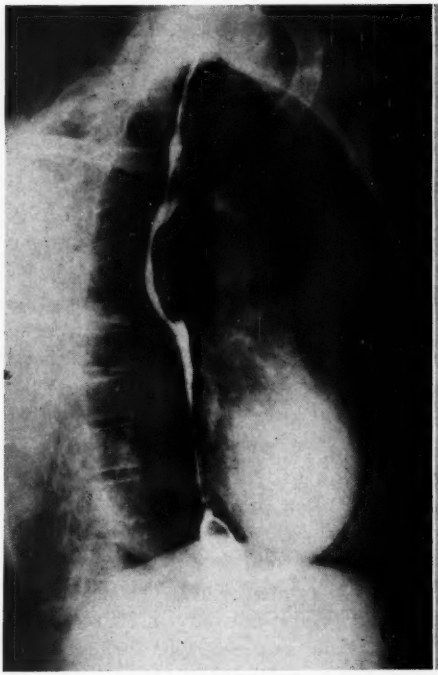

Fig. 1.

Fig. 2.

Fig. 1.—Barium esophagram in right anterior oblique view which shows displacement of esophgus at level of aortic arch.

Fig. 2.—Spot film of lower trachea with patient recumbent, taken following instillation of oily Dionosil. Note pooling in diverticulum.

subjacent tracheocele was excised. In 1951, Clagett[10] operated upon a 52-year-old man who complained of long-standing productive cough. Utilizing a sternum-splitting incision, he resected an acquired diverticulum of the trachea presenting from the left cornu just below the clavicle. Complimentary tracheostomy was performed to reduce the mean intratracheal pressure.

CASE REPORT

R. B., a 59-year-old widow, was seen in consultation because of a chronic recurrent productive cough of 28 years' duration. Prior to age 30, she had enjoyed excellent health with no symptoms referable to the respiratory system. In 1931, about 1 year prior to undergoing a uterine suspension, she had noted the onset of a dry cough, the cause of which was

unexplained. Very shortly following this operation, the cough increased in severity and became productive of mucopurulent sputum. During the ensuing several years the cough was almost constantly present, and severe paroxysms frequently induced vomiting and became quite incapacitating. She sought medical consultations, but repeated physical examinations and chest roentgenograms were said to be unremarkable. Her "chronic bronchitis" was

Fig. 3.

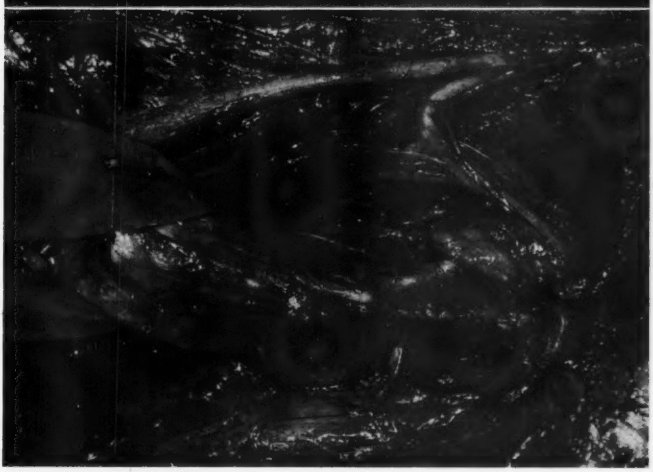

Fig. 4.

Fig. 3.—Photograph at operation with tracheocele open. Tape surrounds the left mainstem bronchus. Note descending aortic arch at right and below.

Fig. 4.—Same view as Fig. 3 which shows closure of membranous trachea. Vagus and recurrent nerves are seen above. Note esophagus below between bronchus and aorta.

treated with a multiplicity of antitussives, sedatives, antihistamines, expectorants, inhalants, and chemotherapeutic agents with only partial or transient relief. In 1948 a severe episode of pneumonitis and pleurisy occurred.

In 1956, while vacationing in Florida, an extraordinarily severe episode of coughing occurred, and she was hospitalized. Chest roentgenograms were said to be normal. Bronchoscopic examination was performed and reported to reveal a "very unusual condition"

characterized by an "impaction of glue-like secretion in the lower trachea." This was aspirated, and the cough tended to subside almost completely for the ensuing 2½ years. However, in March, 1959, it recurred, again becoming productive of thick sputum. She was admitted for study.

Physical examination showed a well-nourished woman with no evidence of dyspnea, cyanosis, or clubbing. There was a hoarse, low-pitched, rattling cough of a very unusual character. Auscultation revealed a loud coarse wheeze over the anterior thorax with maximal intensity over the upper sternum. On inspiration this sounded like swirling eddy currents of air.

Hematologic studies and urinalysis were normal. Sputum smears showed many leukocytes and some bacteria; all cultures showed no growth. An electrocardiogram was normal.

Bronchoscopic examination revealed a cylindrically dilated and somewhat bulbous trachea. Just above the main carina on the posterolateral wall a wide-mouthed orifice was observed communicating with a smooth-lined diverticular pouch. With inspiration the mobile sac wall ballooned out, and at expiration its dome partially prolapsed into tracheal lumen, vibrating against the wall of the orifice.

Conventional roentgenograms of the thorax showed a circumscribed oval radiolucency at the left tracheal border. An esophagram (Fig. 1) showed posterior displacement of the esophageal segment opposite the aortic arch. Tomograms in the coronal plane demonstrated the paratracheal radiolucency to be devoid of lung markings. Angiocardiography showed no abnormality of pulmonary vasculature. Bronchography under fluoroscopic control revealed the following information: with the patient semirecumbent, 4 c.c. of oily Dionosil was instilled by catheter into the mid-trachea and seen to pool in the diverticulum just above the main carina (Fig. 2). The head of the tilt table was then elevated, and the contrast material emptied into both bronchial trees. This occurred in the absence of any coughing; no esophageal dye was seen. The upright films showed moderate cylindrical bronchiectasis of the basilar segments in each lower lobe. Diffuse trachiectasis was also evident.

Left lateral thoracotomy was performed on April 7, 1959, under endotracheal anesthesia. The thin-walled diverticulum presented in the aortic window just under the arch and was dissected free of surrounding tissue. It was quite intimately adherent to the esophagus. The sac measured 3 by 2 by 1.5 cm. Traction sutures were placed, and it was momentarily opened for inspection (Fig. 3). A Satinsky clamp was placed across its base and proximal excision performed, closing the defect with two layers of the silk (Fig. 4). Postoperatively, the patient's course was entirely uneventful. Although still present 3 months later, her cough has become progressively less marked and less productive. The severe paroxysms are no longer present, and subjective improvement is striking.

Microscopic sections of the sac showed layers of membranous trachea with no inflammation.

COMMENT

In this patient the onset of symptoms after the age of 30 and the morphologic characteristics of the tracheocele strongly suggest the latter to have been an acquired lesion. The associated bronchiectasis and trachiectasis lessened our hope for a surgical cure of the cough; however, considerable mitigation of its severity has already occurred.

SUMMARY

The subject of congenital and acquired tracheoceles has been briefly reviewed. A case is reported of a 59-year-old widow with long-standing chronic cough of occult etiology. Excision of a tracheocele was performed with marked palliation.

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ABERRANT LEFT PULMONARY ARTERY

A Correctable Cause of Respiratory Obstruction

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INTRODUCTION

THE SURGICAL RELIEF of respiratory obstruction caused by vascular anomalies was started by Gross[1, 2] in 1945 with division of a double aortic arch. Interest since then has centered around correction of aortic arch anomalies. In the past 2 years we have encountered 3 cases of aberrant left pulmonary artery which caused respiratory obstruction. In these cases, the left pulmonary artery arose on the right of the mid-line and passed posteriorly and to the left between the trachea and esophagus to reach the left hilum (Fig. 1). Since, in 2 of our cases, the surgeon's failure to recognize the true nature of the abnormality led to an incorrect procedure, it seems desirable to call attention to this lesion.

The first case of aberrant left pulmonary artery associated with tracheobronchial obstruction was reported in 1938[3] and 16 years elapsed before the report of 2 additional cases[4, 5] in 1954. A total of 15 cases have now been reported (Table I). The fact that 11 of these have been reported from four clinics suggests that this anomaly is more common than previously appreciated.

CASE REPORTS

CASE I.—(Table I—Case XIII.) L. D., No. 8456, born May 14, 1956, died Sept. 22, 1956. The patient, a white girl, was admitted to the Babies Hospital at the age of 7 weeks with a history of dyspnea since birth. There were wheezes in both lungs and marked stridor. Chest roentgenogram showed obstructive emphysema on the right with mediastinal shift to the left. No abnormalities were seen on direct laryngoscopy or esophagram. Bronchoscopy was not done.

At the age of 11 weeks, with a presumptive diagnosis of bronchogenic cyst, the right pleural cavity was opened. The right lung was found to be emphysematous, but no cyst or other abnormality causing bronchial obstruction was found. The mediastinum was not dissected.

Following operation, stridor and tachypnea persisted. A combined tracheo-esophagram showed marked indentation of the posterior trachea and anterior esophagus at the level of the carina, well visualized in the lateral view but poorly seen in the posteroanterior projection (Fig. 2).

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Two weeks later, the right chest was re-opened and the mediastinum dissected. A large blood vessel was found arising from the right pulmonary artery anterior to the bronchus, passing posteriorly over the origin of the right primary bronchus around the lateral aspect of the trachea, and then medially to the left between the trachea and esophagus where its further course could not be identified. The vessel was thought to be a right patent ductus

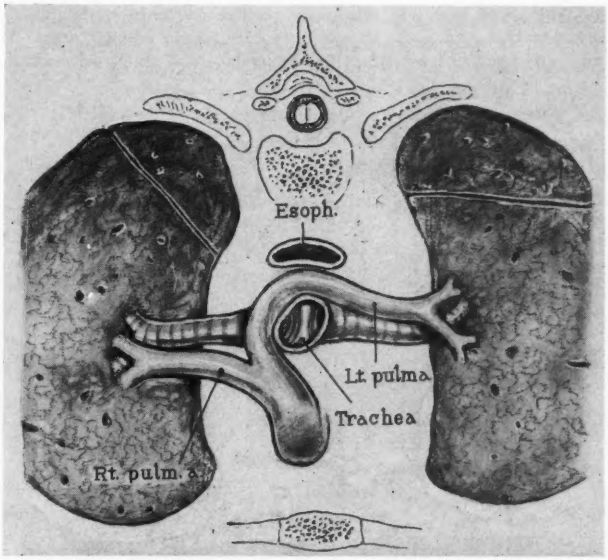
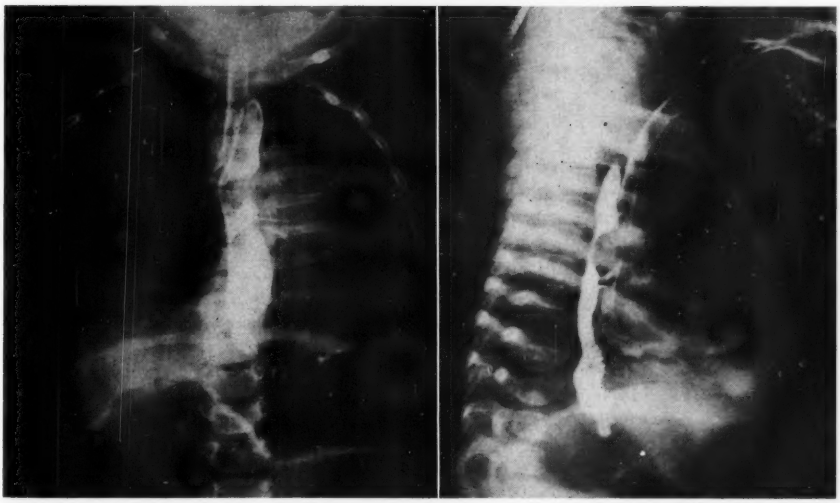


Fig. 1.—Diagrammatic representation of aberrant left pulmonary artery (cross section).



A. *B.*

Fig. 2.—Case I. *A*, Posteroanterior tracheo-esophagram which fails to show the abnormality. *B*, Lateral tracheo-esophagram demonstrating the aberrant left pulmonary artery.

arteriosus. After 5 minutes of well-tolerated occlusion it was divided. The postoperative course was characterized by increasing respiratory distress, and death occurred 48 hours later. Postmortem examination revealed, in addition to the aberrant left pulmonary artery (Fig. 3) which had caused compression of the trachea and right main bronchus, the following pathologic features: (1) persistent left superior vena cava emptying into a large coronary sinus, (2) ostium secundum defect 8 mm. in length (slit-like), (3) anomalous branch of left pulmonary artery to right upper lobe, (4) anomalous tracheal cartilages: complete cartilaginous rings in the lower trachea and main bronchi, (5) high origin of right upper lobe bronchus (epiarterial), and (6) emphysema in the right upper lobe and atelectasis in the right middle and lower lobes.

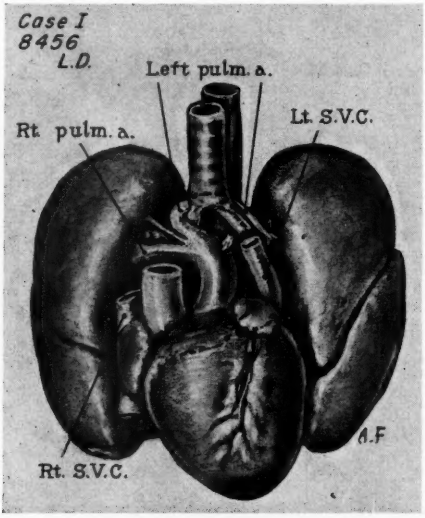


Fig. 3.—Case I (Table I—Case XIII).

CASE II.—(Table I—Case XIV.) D. A. No. 8678, born Dec. 13, 1952, died Nov. 15, 1957.

The patient, a Negro girl, was first admitted to the Babies Hospital at the age of 2 weeks with a history of respiratory distress since birth. Stridor and moderate tachypnea had persisted with frequent exacerbations throughout her life. At the age of 6 weeks the diagnosis of congenital heart disease was made on the basis of a harsh systolic murmur, cyanosis, and an abnormal cardiac configuration on a chest roentgenogram. Cardiomegaly was progressive. An esophagram at the age of 8 months disclosed a right aortic arch without other evidence of esophageal compression.

Angiocardiography done at this time was interpreted as showing persistent truncus arteriosus. The course of the pulmonary arteries was not clear. Bronchoscopy and tracheobronchogram were not done. After repeated exacerbations of symptoms, she was admitted for the eighth time at the age of 4 years and 11 months in severe congestive heart failure and shock. Death occurred 4 hours later. Necropsy (Fig. 4) revealed in addition to the aberrant pulmonary artery: (1) tetralogy of Fallot with atresia of the pulmonary valve and high ventricular septal defect (1.3 cm.), (2) ostium secundum defect (0.8 by 1.5 cm.), (3) patent ductus arteriosus (1.2 cm. circumference), (4) peristent left superior vena cava entering the coronary sinus, (5) right aortic arch and anomalous origin of the right common carotid (right vertebral and right subclavian arteries), (6) singled-lobed left lung, (7) anomalous branch of the left pulmonary artery supplying the right upper lobe, and (8) hyperostosis of calvarium with compression of frontal lobes.

CASE III.—(Table I—Case XV.) N. F., No. 8888, born Oct. 19, 1958, died Dec. 24, 1958.

This infant was admitted to the Babies Hospital at the age of 8 weeks with a history of wheezing for 3 weeks. Physical examination revealed stridor, chest retraction, and slight cyanosis. Chest roentgenogram showed emphysema of the right lung. Esophagram disclosed anterior indentation at the level of the carina, best seen in the left anterior oblique view. The child's course was characterized by rapidly progressive respiratory distress. A tracheoesophagram was done by the "gulp" technique (described later) which showed separation of the trachea and esophagus by an abnormal structure, well seen in the posteroanterior roentgenogram (Fig. 5, *A*). A left anterior oblique esophagram demonstrates the size of the vessel wall (Fig. 5, *B*). A presumptive diagnosis of bronchogenic cyst was made. At operation the right lung was found to be almost completely consolidated. An abnormal vessel was

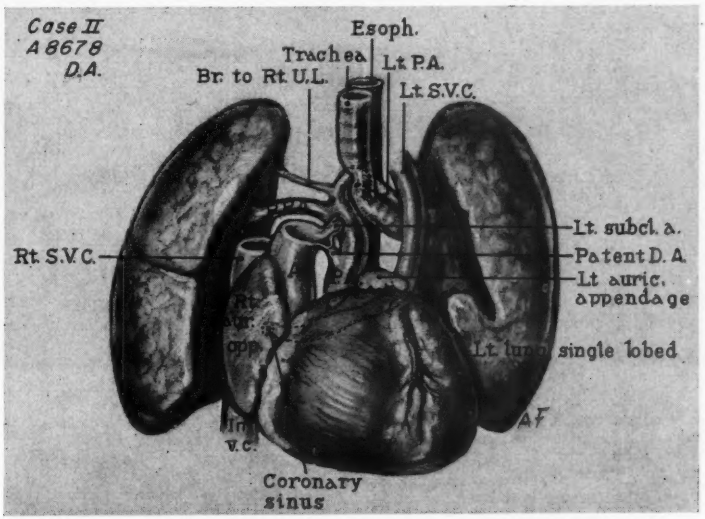
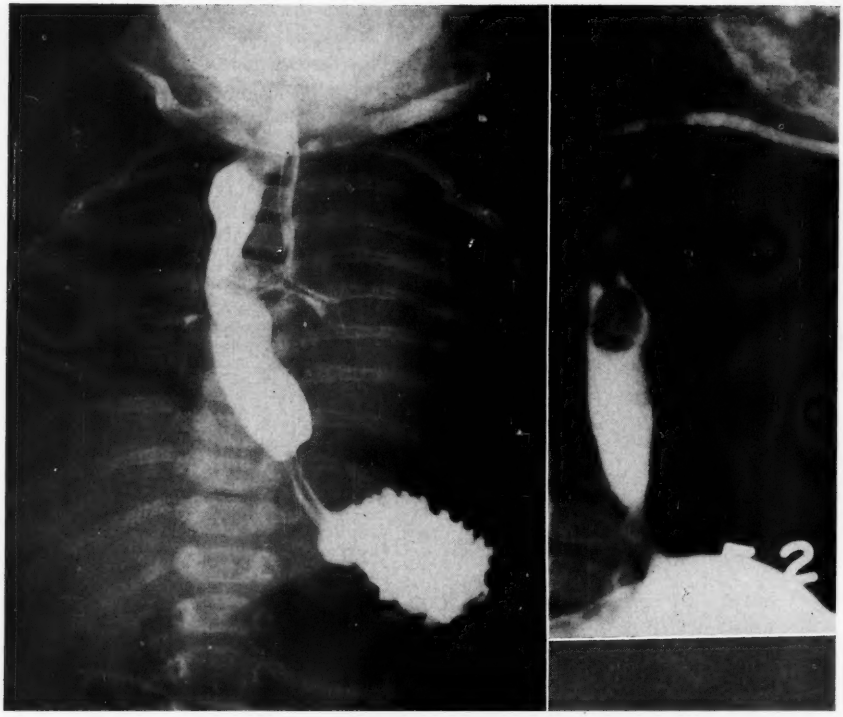


Fig. 4.—Case II (Table I—Case XIV).

identified arising to the right of the mid-line in the mediastinum, anterior to the trachea, passing to the right around the lateral and posterior aspects of the trachea, and then medially to the left, anterior to the esophagus. The possibility that this represented an aberrant left pulmonary artery was considered but aspiration of the vessel yielded bright red blood under arterial pressure. A diagnosis of double aortic arch was made. After occlusion for 5 minutes was well tolerated, the vessel was divided. Postoperatively, assisted respiration was required, and heart action ceased after 6 hours. Necropsy examination revealed, in addition to the aberrant pulmonary artery (Fig. 6) with compression and malacia of the right main-stem bronchus, the following pathologic features: (1) ostium secundum defect 5 by 11 mm. and fenestrated septum primum with its largest opening 4 by 6 mm., (2) patent ductus arteriosus, 7 mm. in length and 4 mm. in circumference, (3) anomalous tracheal cartilages (complete cartilaginous rings in the lower trachea and main bronchi), and (4) absence of gall bladder.

DISCUSSION

The diagnosis of aberrant left pulmonary artery should be considered in all infants with persistent stridor. Symptoms of respiratory distress appeared in the first month of life in all but one reported case, with stridor generally noted



A. *B.*

Fig. 5.—Case III. *A*, Posteroanterior tracheo-esophagram which demonstrates separation of the trachea and esophagus by the aberrant left pulmonary artery.

B, Left anterior oblique esophagram which shows esophageal indentation by the aberrant left pulmonary artery.

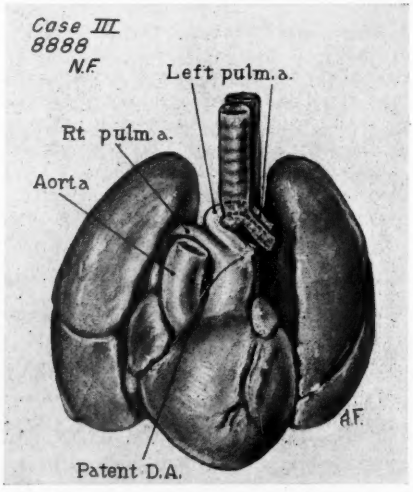


Fig. 6.—Case III (Table I—Case XV).

at birth or shortly afterward. In contrast to the clinical picture presented by most other vascular ring anomalies, symptoms of esophageal obstruction have not been noted. Patients have presented with any or all of the following symptoms: stridor, dyspnea, cyanosis, and repeated respiratory infections. The course is usually progressive with occasional lessening but never disappearance of symptoms. Physical findings may include stridor, retraction, tachypnea, wheezing, hyperresonance on the right with tracheal deviation to the left, and cyanosis. When pulmonary infection is superimposed, bilateral râles, rhonchi, and fever may appear. This can obscure the primary factor of respiratory obstruction.

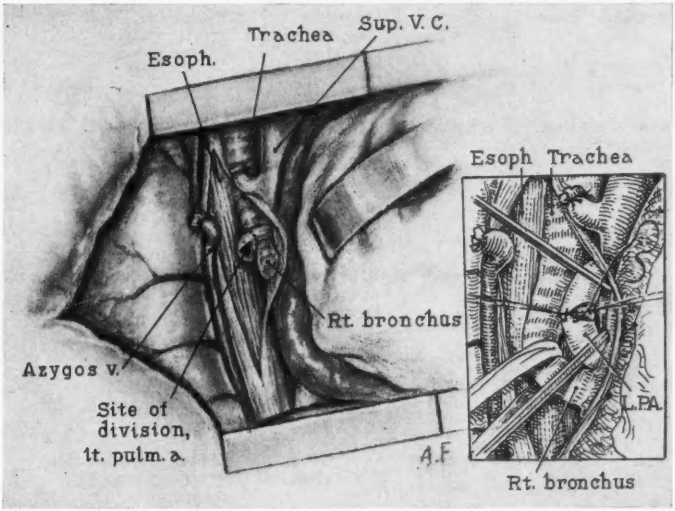


Fig. 7.—Surgical approach for correction of aberrant left pulmonary artery. Insert shows the divided left pulmonary artery being re-anastomosed after division and transplantation medial to the right main-stem bronchus.

Chest x-ray findings have been variable. The most common picture is that of emphysema of the right lung with mediastinal shift to the left. Lateral view may show narrowing of the lower trachea. Bronchoscopy has been consistently useful in demonstrating narrowing of the trachea or right main-stem bronchus. However, this procedure is hazardous in infancy and it is our belief that the diagnosis can be made conclusively by other techniques. Angiocardiography has been reported as diagnostic.[8] In Case II of the present series, the anomalous left pulmonary artery was not visualized by this technique because of the presence of a tetralogy of Fallot with atresia of the pulmonary valve.

In our experience, the most important diagnostic aid has been the combined tracheo-esophagram, utilizing the "gulp" technique. In this procedure, 5 to 10 c.c. of aqueous Dionosil is instilled into the posterior pharynx followed immediately by occlusion of the nose and mouth. This causes the patient to swallow

TABLE I

CASE NO.	AUTHOR	SEX	AGE AT ONSET OF SYMPTOMS	DIAGNOSTIC TESTS			ANGIO
				X-RAY CHEST	ESOPHAGRAM	BRONCHOSCOPY	
I.	Scheid[3]	Male	7 mo.	Normal	—	Narrowing of lower trachea	
II.	Welsh & Munro[4]	Male	2 wk.	Normal	Unsatisfactory study	—	
III.	Potts et al.[5]	Male	Birth	Emphysema rt. lung; left mediastinal shift	Normal	Extrinsic pressure on lower trachea and rt. main bronchus	
IV.	Morse & Gladding[6]	Male	Birth	Changing findings rt. lung; left mediastinal shift	—	Narrowing rt. mainstem bronchus	
V.	Wittenborg et al.[7]	Male	4 da.	Diffuse emphysema; narrowing trachea	Posterior indentation; slight anterior indentation	Narrowing trachea just above carina	
VI.		Male	Birth	Normal	—	—	
VII.	Hiller & Maclean[8]	Female	Birth	—	Anterior indentation of esophagram	—	Demon erran nary
VIII.		Male	?	—	Anterior indentation of esophagram	—	Demon erran nary
IX.		Female	Birth	—	Anterior indentation of esophagram	—	Demon erran nary
X.	Contro et al.[9]	Female	1 mo.	? Enlarged thymus	Anterior indentation esophagus	Moderate narrowing of distal trachea; collapsed rt. mainstem bronchus (performed on operating room table with chest open)	
XI.		Male	4 da.	Normal	—		

ANGIOCARDIOGRAM	AGE AT OPERATION	OPERATION	AGE AT DEATH	DIAGNOSIS ANTE MORTEM	ADDITIONAL FINDINGS AT AUTOPSY
—	—	None	10 mo.	No	Complete tracheal cartilaginous rings; patent ductus arteriosus; ostium secundum defect (1 by 1.5 cm.)
—	6 mo.	Anomaly appreciated at exploration; cardiac arrest before correction attempted	6 mo.	At operation	None mentioned
—	5 mo.	Anomalous left pulmonary artery divided and re-anastomosed to the left of the trachea; excellent result	—	Yes	—
—	1 mo.	Anomalous left pulmonary artery divided and re-anastomosed medial to the trachea; initial improvement with early recurrence of respiratory obstruction	1½ mo.	At operation	None
—	—	None	3 mo.	No	Aberrant right subclavian artery; persistent left superior vena cava to coronary sinus; hypoplasia left kidney; partial malrotation on intestine; hemivertebrae
—	—	None	3 mo.	No	Ventricular septal defect; dextroposition of aorta; coarctation of aorta
Demonstrated aberrant left pulmonary artery	2½ yr.	Anomalous left pulmonary artery divided and re-anastomosed to the left of the trachea; postoperative angiocardiogram showed no filling of left pulmonary artery; stridor relieved	—	Yes	—
Demonstrated aberrant left pulmonary artery	2 yr.	Anomalous left pulmonary artery re-anastomosed medial to the trachea; postoperative cardiac arrest	2 yr.	Yes	No other cardiovascular anomalies
Demonstrated aberrant left pulmonary artery	—	None	—	Yes	—
—	2½ mo.	Left exploratory thoracotomy performed; anomalous left pulmonary artery not recognized	2½ mo.	No	None mentioned
	—	Repair of imperforate anus	4 da.	No	Repaired imperforate anus; harelip; cleft palate; persistent left superior vena cava into coronary sinus; atrioventricularis communis

TABLE I. CON

CASE NO.	AUTHOR	SEX	AGE AT ONSET OF SYMPTOMS	DIAGNOSTIC TESTS		
				X-RAY CHEST	ESOPHAGRAM	BRONCHOSCOPY
XII.	Sherman[10]	Male	Birth	Emphysema rt. lung; left mediastinal shift	—	—
XIII.	Present report	Female	Birth	Emphysema rt. lung; left mediastinal shift	Lateral indentation esophagus	Tracheo-bronchogram; no contrast media into rt. main bronchus
XIV.	Present report	Female	Birth	Cardiomegaly	Normal	—
XV.	Present report	Male	1 mo.	Increased density rt. base	Anterior indentation esophagus	Tracheo-bronchogram; separation of trachea and esophagus

and aspirate, allowing simultaneous visualization of the esophagus and tracheobronchial tree. The aberrant pulmonary artery is well outlined by the resultant filling defects in the posterior trachea and anterior esophagus. It has been stated that the diagnostic identation of the esophagus is best seen in the lateral projection.[7] Our cases have been variable in this regard. In Case I, the esophagus appeared normal in the posteroanterior film while the lateral view showed deep anterior indentation; in Case III, the reverse was true. Ideally, posteroanterior, lateral, and oblique projections should be taken with radiopaque material in the trachea and esophagus. It should be emphasized that virtually the only other vessel which passes between the trachea and esophagus is an aberrant subclavian artery which, in 15 per cent of cases, lies between these two structures.[11] In this condition, however, the indentation of the esophagus is oblique in the posteroanterior projection and is seen at a higher level.

In cases of aberrant left pulmonary artery, the outstanding pathologic feature has been the uniformity of the anomaly. In each case it has consisted of an elongated main pulmonary artery segment with the left branch arising well in the right mediastinum and passing posteriorly between the trachea and esophagus to the left hilum. Either the trachea, the right main-stem bronchus or both may be obstructed as a result (see Fig. 1). The term "vascular sling"[9] has been proposed to differentiate this condition from the more common vascular ring anomalies.

CONT'D

ANGIOCARDIOGRAM	AGE AT OPERATION	OPERATION	AGE AT DEATH	DIAGNOSIS ANTE MORTEM	ADDITIONAL FINDINGS AT AUTOPSY
—	3 wk.	Right middle and lower lobe lobectomies for obstructive emphysema; anomaly of pulmonary artery not recognized	6 mo.	No	Bilateral obstructive emphysema; rt. subclavian and rt. and left common carotids arose from innominate artery
—	11 wk.	Right exploratory thoracotomy; anomaly of pulmonary artery not recognized	4 mo.	No	Complete cartilaginous rings trachea; rt. upper lobe artery from left pulmonary artery; persistent left superior vena cava entering coronary sinus; ostium secundum defect; epiarterial right upper lobe bronchus
	14 wk.	Right exploratory thoracotomy; anomalous pulmonary artery thought to be a patent ductus and ligated			
Did not demonstrate anomalous pulmonary artery	—	None	4 yr., 11 mo.	No	Tetralogy of Fallot with pulmonary atresia; ostium secundum defect; right aortic arch; patent ductus arteriosus; persistent left superior vena cava into coronary sinus; single lobed left lung; rt. upper lobe artery from left pulmonary artery
—	2 mo.	Right exploratory thoracotomy; anomalous pulmonary artery thought to be anomalous aortic arch and ligated	2 mo.	No	Ostium secundum defect; patent ductus arteriosus; cartilaginous rings; absence of gall bladder

Associated congenital anomalies are noted in Table I. Of the 12 cases examined postmortem, complete tracheal cartilaginous rings were present in 3, while 4 patients had persistent left superior vena cava. Four patients (Cases VI, XI, XIV, XV in Table I) had congenital cardiac anomalies, while 2 additional infants (Cases V, XII) had anomalies of the systemic arterial system.

The embryologic origin of this defect is unknown. The development of the left pulmonary artery from other than the usual channels of the primitive vascular plexus, which is the forerunner of the aortic arches, might lead to such an anomaly. If this is true, the malformation dates from the fifth or sixth week of fetal life. Many of the associated anomalies listed in Table I, including vena caval anomalies, cleft palate, and imperforate anus, probably date from the same stage of development.

Surgical correction of this anomaly is achieved by dividing the aberrant pulmonary artery and re-anastomosing it in front of the trachea. This was done successfully in 2 of the 8 cases in which operation was performed (III and VII —Table I). Improvement in preoperative diagnosis and better understanding of the anomaly at the operating table should increase the success rate markedly.

A right anterolateral approach gives the optimum exposure. The azygos vein and pleura must be divided along the tracheoesophageal groove. Failure to do this has resulted in failure to see the anomaly in several cases. Fig. 7 shows the vessel as seen from this approach. The point of origin from the main

pulmonary artery cannot be visualized in this limited dissection, consequently the vessel has been mistaken for part of an aortic ring anomaly. This should not be a source of confusion. To our knowledge, virtually the only other vessel which passes between the trachea and the esophagus is an aberrant subclavian artery[11]; this vessel does not encircle the trachea and is at a higher level.

In Case III, high pressure and the aspiration of arterial blood led to the division of the vessel with the presumptive diagnosis of double aortic arch. The explanation for this became apparent at postmortem examination. A patent ductus arteriosus emptied from the aorta into the main pulmonary artery just proximal to the origin of the aberrant left pulmonary artery. A large secundum atrial septal defect was also present, perhaps permitting additional right-to-left shunt.

The anastomosis must be done with great rapidity since the right lung is often compromised. Hypothermia might offer additional protection during performance of the anastomosis.

SUMMARY

Aberrant left pulmonary artery should be considered in the differential diagnosis of all infants with persistent stridor. In this condition, the left pulmonary artery arises in the right mediastinum and passes posteriorly between the trachea and esophagus to the left lung, resulting in obstruction of the trachea or right main-stem bronchus.

Fifteen cases of this anomaly are summarized. Diagnosis can be readily established by combined tracheo-esophagram. Surgical correction is discussed.

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A COMPARATIVE STUDY OF HEPARIN NEUTRALIZATION AND THE DANGER OF BLOOD CLOTTING ABNORMALITIES WITH PROTAMINE AND POLYBRENE

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THE RAPID INCREASE in use of extracorporeal circulation has emphasized the need for optimum control of anticoagulation and coagulation of blood. Although protamine sulfate will completely neutralize heparin, it has a specific anticoagulant action of its own and has been incriminated as a possible cause of clotting abnormalities after perfusion[1, 4]; a certain incidence of undesirable systemic reactions has also been recorded. A drug with equal heparin-neutralizing properties, but which could be used with greater safety, would be highly desirable. Polybrene,† a polymeric quaternary ammonium salt, has recently been proposed as such an agent and studies have been performed indicating that it will produce satisfactory neutralization of heparin[5, 7]; however, data on its in vivo anticoagulant activity have not been presented, and the quantitative aspects of heparin neutralization in comparison with protamine have not been well defined.

In a series of animal experiments, Polybrene and protamine sulfate were compared with respect to effectiveness of heparin neutralization and neutralizing dosage levels, and quantitative studies were performed of in vivo anticoagulant effect of each drug at dosage levels within the range commonly employed clinically.

METHODS

Adult mongrel dogs were lightly anesthetized with intravenous Pentothal and plastic cannulae were introduced through the femoral veins into the inferior vena cava for blood sampling. Before collection of each sample the cannulae were flushed by preliminary withdrawal of blood.

Heparin neutralization was estimated by titrations[1] using protamine or Polybrene, respectively, and the clotting time was measured simultaneously

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†Polybrene (hexamethrine bromide) supplied by Abbott Laboratories, Chicago, Ill.

with each titration by a three-tube Lee-White technique. The titration was used to indicate the relative degree of neutralization of heparin or excess of the neutralizing agent and the coagulation time was used as a more precise quantitative measure of the clotting time.

DESIGN OF EXPERIMENTS

Adult mongrel dogs in pairs of similar weight were selected at random. Experiments were performed using Polybrene in one dog of each pair and protamine* in the other, with each of the stages of the experiments carried out at identical times. This technique seemed satisfactory from the standpoint of providing comparative information on the relative effect of the two drugs at risk.

Studies were performed in 20 animals with protamine and in a concomitant group of 20 animals with Polybrene. In each animal, a control titration was performed and clotting time determined and then heparin,† 2.5 mg. per kilogram, was administered intravenously. Fifteen minutes after the administration of heparin, the titration was repeated and coagulation time determined, followed immediately by the intravenous administration of a predetermined amount of neutralizing agent. At subsequent 15-minute intervals, repeat titrations were performed and coagulation times determined and additional amounts of the neutralizing agents were given until a total dose equal to 200 per cent of the original heparin dose by weight was reached. In each pair of experiments, identical amounts of protamine and Polybrene were given at each stage, calculated on a body-weight basis.

RESULTS

The qualitative results in each group are given in Tables I and II. Significant inhibition of clotting by the heparin-neutralizing agent was considered to be present only in those animals in which titration indicated an excess and the clotting time was increased by 5 minutes or more above either the control level or the lowest post-neutralization level. For this evaluation a clotting time within 5 minutes of control levels was considered as demonstrating satisfactory neutralization. By this standard, 12 of 20 dogs in which heparin was neutralized by protamine showed inhibition of clotting due to protamine. Inhibition occurred chiefly at dosage levels of 150 to 200 per cent of the original heparin dose by weight. In the Polybrene-treated dogs, inhibition of clotting occurred in 11 of 20 dogs, with the majority of instances of inhibition occurring at the 150 per cent and 200 per cent dosage levels. There was no significant difference between protamine and Polybrene in the incidence of inhibition of coagulation.

*Protamine sulfate injection, 1 per cent, was used in all experiments. (Supplied by Eli Lilly Co., Indianapolis, Ind.)

†Heparin sodium, 1,000 units per cubic centimeter (10 mg.), was used in all experiments. (Supplied by Abbott Laboratories, Chicago, Ill.)

TABLE I*

EXPERIMENT NUMBER	POLYBRENE DOSAGE IN PER CENT OF HEPARIN GIVEN						
	50%	75%	90%	100%	125%	150%	200%
1	0			0		+	+
2	—			0		0	0
3		0				0	0
4		0				0	0
5		0				+	+
6			+			+	+
7			0			0	0
8			0			+	+
9				0		0	0
10				0		0	0
11				0		+	+
12				0		0	+
13				+		+	+
14				0		0	+
15					0		0
16					0		+
17					0		0
18						+	+
19						+	+
20						0	0
Totals							
–	1						
0	1	3	2	7	3	9	9
+			1	1		8	11

*(–) indicates heparin excess as determined by titration. (0) indicates complete neutralization of heparin with clotting time essentially normal. (+) indicates protamine excess with clotting time prolonged more than 5 minutes above control or lowest post-neutralization time.

TABLE II*

EXPERIMENT NUMBER	PROTAMINE DOSAGE IN PER CENT OF HEPARIN GIVEN						
	50%	75%	90%	100%	125%	150%	200%
1	—			+		+	+
2	0					0	0
3		0		+		+	+
4		0				0	+
5		0				0	0
6			0			+	+
7			0			+	+
8			0			0	0
9				0		0	0
10				0		+	+
11				0		0	0
12				0		+	+
13				0		+	+
14				0		+	+
15					0		+
16					0		0
17					0		0
18						0	+
19						0	+
20						0	0
Totals							
–	1						
0	1	3	3	6	3	9	8
+				2		8	12

*(–) indicates heparin excess as determined by titration. (0) indicates complete neutralization of heparin with clotting time essentially normal. (+) indicates protamine excess with clotting time or lowest post-neutralization time.

The mean increase in clotting time above optimum levels, as determined by the three-tube coagulation time, was 5.5 minutes in the group treated with protamine and 7.7 minutes in the group receiving Polybrene (Table III). In each case this is a significant increase in coagulation time as compared to control levels ($0.01 > P > 0.001$). However, the difference between protamine and Polybrene is not significant ($0.40 > P > 0.30$). Protamine did not produce prolongation of clotting times of the magnitude seen in several experiments with Polybrene (Experiments 6, 13, 18).

TABLE III. INCREASE IN CLOTTING TIME ABOVE CONTROL LEVELS (IN MINUTES)*

EXPERIMENT NUMBER	PROTAMINE	POLYBRENE
1	+10	+ 7.5
2	0	0
3	+16	0
4	+ 6.5	0
5	0	+ 8
6	+18	+29
7	+ 6.5	0
8	0	+ 8.5
9	0	0
10	+ 5.5	0
11	0	+ 7
12	+15	+13.5
13	+ 7.5	+26.5
14	+ 5.0	+ 7.0
15	+ 6.5	0.0
16	0.0	+14.0
17	0.0	0.0
18	+ 5.0	+19.0
19	+10.0	+15.0
20	0.0	0.0
Mean	5.5	7.7
S. D.	7.7	11.5
S. D. Mean	1.73	2.6

*In each instance the maximum increase in clotting time above control levels is recorded. This increase usually, but not always, occurred at the maximum level of protamine administration.

With each drug, dosages of 75 per cent or more by weight of the original heparin doses consistently produced complete neutralization of the heparin and restored the clotting time to normal.

DISCUSSION

A close examination of data in published studies on the neutralization of heparin by protamine or Polybrene indicates that it is not uncommon for the clotting time to fail to return entirely to normal levels after apparently adequate neutralization. Abnormal coagulation of the blood has also been noted frequently after neutralization of heparin following total body perfusion with an artificial pump-oxygenator, even though titration tests eliminate inadequate heparin neutralization as a cause for the observed abnormality in clotting.

Although the potential danger of protamine as a specific anticoagulant has been mentioned as a possible cause of this abnormality,[1, 4] quantitative in

vivo studies of this potential hazard have not been presented. Similarly, although Polybrene has a known anticoagulant effect in vitro,[6] there has been no quantitative data from in vivo studies. Previous experimental studies[1] on the activity of heparin and aspects of protamine neutralization have demonstrated that it is not uncommon to have protamine inhibition of clotting after administration of doses comparable to those used clinically. Although the resultant defect in coagulation is ordinarily small, it may be great enough to produce abnormal bleeding.

The present studies and others[1, 2, 3] suggest that the commonly used neutralizing dosages of 100 per cent to 200 per cent by weight of the heparin given may be unnecessarily large and, in fact, hazardous. Further, in these studies the initial neutralizing dose was given only 15 minutes after administration of heparin; with the passage of time there is a rapid decrease in the active heparin level, so that progressively smaller amounts of neutralizing drugs are required to restore normal blood clotting.[1]

In our initial experimental experience with Polybrene, it appeared that a more normal coagulation time was obtained than with protamine. This observation seems to be explained by the smaller amounts of Polybrene which we used, 0.7 to 1.0 times the amount of heparin given. Since the heparin-neutralizing properties of the two drugs are approximately equal by weight, use of a larger dose with one or the other will tend to produce a proportionately greater incidence of inhibition of clotting due to excess. The data obtained in these comparative experiments do not indicate any significant difference between Polybrene and protamine, either in effectiveness of heparin neutralization or in tendency to produce inhibition of blood clotting with excessive doses.

Although these data may not be directly applicable to the post-perfusion state, they suggest that a more cautious use of these drugs in neutralizing heparin after extracorporeal circulation may lessen the incidence of unexplained abnormalities in bleeding and clotting.

SUMMARY

1. A comparative experimental study of in vivo heparin neutralization and production of inhibition of blood clotting by protamine and Polybrene was carried out in dogs.

2. Dosages of 75 per cent or more by weight of the original heparin dose were consistently sufficient to produce complete heparin neutralization with either protamine or Polybrene.

3. Both protamine and Polybrene produced significant elevation of coagulation time above control levels in over 50 per cent of these experiments. The abnormalities of clotting occurred chiefly at dosages of 150 to 200 per cent of the heparin dose by weight, well within the range in common clinical use for heparin neutralization. This dosage ratio is unnecessarily large for optimal neutralization.

4. The increase in coagulation time produced by these drugs in the dosages used were small compared to that produced by heparin. Nevertheless, it may be sufficient to cause significant bleeding.

5. No definite superiority of either drug was evident in the parameters tested.

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THE EFFECT OF 2 PER CENT CARBON DIOXIDE ON pH AND pCO_2 DURING EXTRACORPOREAL CIRCULATION

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CARBON DIOXIDE is routinely added to artificial oxygenators during extracorporeal perfusion, and yet, there is no evidence to indicate that this is necessary. Björk[1] observed an uncompensated carbon dioxide deficit with an increase in pH when the brain alone was perfused with 100 per cent oxygen in the oxygenator. Stokes and Gibbon[2] showed similar changes during recirculation prior to perfusion. However, they also observed that when the animal's metabolic carbon dioxide had to be removed, the use of 100 per cent oxygen proved necessary to wash out this carbon dioxide and keep the pH within a normal physiologic range. Mendelsohn and his associates[3] warned against excessive "carbon dioxide wash out" during perfusion with 100 per cent oxygen.

We, on the other hand, observed in the laboratory that prolonged perfusion utilizing 100 per cent oxygen in the oxygenator did not result in this so-called "carbon dioxide wash out." Therefore, we elected to omit carbon dioxide in a series of patients and compared the results from this group with the results obtained in patients in whom 2 per cent carbon dioxide was added to the oxygenator.

METHODS

Data were obtained from 56 patients undergoing 57 separate perfusions. In 35 perfusions, 100 per cent oxygen was passed through the oxygenator; in the remaining 22 cases, a mixture of 98 per cent oxygen and 2 per cent carbon dioxide was used at flow rates of 8 to 10 L. per minute. All patients were perfused with a rotating disc oxygenator.[4] Duration of total cardiopulmonary bypass ranged from 9 to 92 minutes. Flow rates varied between 50 c.c. per kilogram per minute (adults) and 140 c.c. per kilogram per minute (small children) and averaged 80 c.c. per kilogram per minute. The ages of the patients varied from 2.5 to 61 years and their weight from 12.2 to 80.05 kilograms.

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The blood was initially oxygenated with room air by slowly revolving the discs through the priming volume in the oxygenator. No gases were passed through the oxygenator until just prior to the start of perfusion. In this way, low initial carbon dioxide tension in the extracorporeal circuit was avoided.

Arterial and venous blood samples were obtained from the extracorporeal system at the start and finish of the period of total cardiopulmonary bypass. In addition, pre-perfusion samples were obtained from 18 patients. Samples were kept under oil in a buffered fluoride solution at 4° C. until analyzed within a few hours. Oxygen and total carbon dioxide were measured after Van Slyke and Neill.[5] A Cambridge Research Model pH meter was used to measure blood pH. All values were corrected to 37° C. Blood pCO_2 was calculated from pH and total carbon dioxide according to the nomogram of Van Slyke and Sendroy.[6] The electroencephalogram and electrocardiogram were monitored continuously during perfusion.

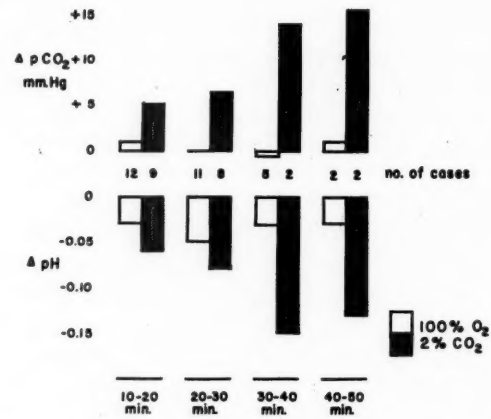

Fig. 1.—Change in pH and pCO_2 as related to perfusion time.

RESULTS

Data relating to perfusion are summarized in Table I. The average fall in arterial pH during perfusion in the cases in which 100 per cent oxygen was employed was from 7.39 to 7.35 while pCO_2 increased slightly from 23.8 to 24.1 mm. Hg. In the second group in which 2 per cent carbon dioxide was added during perfusion, the arterial pH fell from 7.32 to 7.23 at the same time that the pCO_2 rose from 31.3 to 38.6 mm. Hg. The higher initial value of pCO_2 observed in this second group is explained by the fact that extra carbon dioxide had been flowing into the oxygenator for several minutes during the period of partial perfusion and before the blood samples were obtained. There was a significant difference in the changes in these two groups.

The effect of duration of perfusion is shown in Fig. 1. When no carbon dioxide was added, lengthening perfusion did not result in a fall in pCO_2 or in a progressive fall in pH. In contrast, when carbon dioxide was added, increasing perfusion time up to 50 minutes resulted in an increase in pCO_2 and

a decrease in pH. In 3 cases, not included in this figure, in which perfusion lasted over an hour and 100 per cent oxygen was used, there was no significant change in either pH or pCO_2.

The relation between pH and pCO_2 in the final arterial sample is shown in Fig. 2. In 30 of the 35 perfusions in which no carbon dioxide was added, the pH was greater than 7.30 and the pCO_2 was less than 30 mm. Hg in all but one. Four patients in this group showed a final pCO_2 of less than 20 mm. Hg. All 4 patients, however, started with a low pCO_2 due to hyperventilation by the anesthesiologist prior to perfusion and all 4 survived without complications. In all 35 cases, the electroencephalogram remained normal. In 19 of the 22 perfusions in which 2 per cent carbon dioxide was added during perfusion, the pH fell below 7.30. In all 22 cases the pCO_2 was greater than 30 mm. Hg.

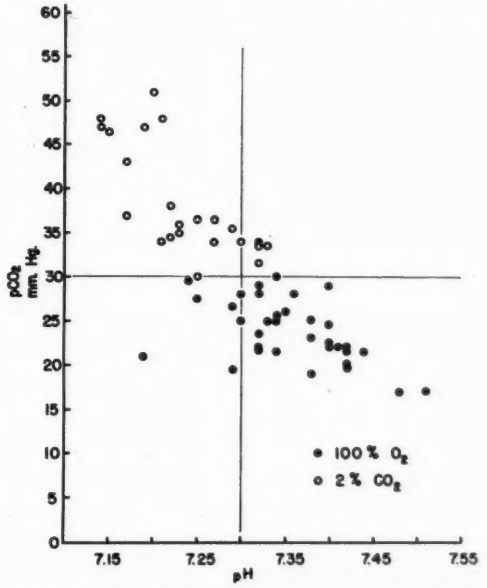

Fig. 2.—Relation between arterial pH and pCO_2 at the finish of perfusion.

In patients in whom a respiratory alkalosis was created by hyperventilation before perfusion, the pH tended to return to normal values, but the pCO_2 remained low if 100 per cent oxygen was used. If carbon dioxide was added, the pH fell as the carbon dioxide rose.

TABLE I. AVERAGE CHANGE IN pH AND pCO_2 DURING EXTRACORPOREAL CIRCULATION

		START	FINISH	CHANGE*	SIGNIFICANCE
pH	100% O_2	7.39	7.35	−0.04 ± 0.04	$p < 0.003$
	2% CO_2	7.32	7.23	−0.09 ± 0.06	
pCO_2	100% O_2	23.8	24.1	+0.3 ± 4.2	$p < 0.001$
	2% CO_2	31.8	38.6	+7.3 ± 5.3	

*Mean ± standard deviation.

DISCUSSION

Using the same oxygenator as Mendelsohn and his co-workers,[3] we have not been able to confirm their observation that "if carbon dioxide is not added to the oxygenator, a marked respiratory alkalosis occurs, with carbon dioxide 'washout'."[7] To the contrary, our observations in patients show that carbon dioxide tension in arterial blood is maintained at nearly constant levels when carbon dioxide is omitted. DeWall,[8] Helmsworth[9] and their co-workers have also observed minimal change in pCO_2 during perfusion. The slight fall in pH in the patients in whom 100 per cent oxygen was used is consistent with the known development of metabolic acidosis that occurs during perfusion, irrespective of the type of oxygenator employed.[3, 8, 10, 11, 12] That this fall in pH was minimal in these cases is probably related to the high perfusion rates used in this series. Addition of carbon dioxide during perfusion does not compensate for this metabolic acidosis but, by superimposing a respiratory acidosis depresses pH even further.

The constancy of the arterial pCO_2 when 100 per cent oxygen is used is difficult to explain. Mechanical factors of the oxygenator can be excluded since no change in pCO_2 was noted whether perfusion was maintained at 1,200 or 2,600 c.c. per minute or for 9 or 60 minutes with the same sized oxygenator at the same oxygen flow rate. The possibility arises, therefore, that there is an intrinsic mechanism in the blood buffer systems (hemoglobin, plasma proteins, phosphate, etc.) that maintains carbon dioxide tension under the conditions of total body perfusion.

The low pCO_2 observed in some of our patients and also in those reported by Mendelsohn[3] was due to hyperventilation by the anesthesiologist prior to perfusion. Carbon dioxide tension can be maintained by controlling repiration, avoiding hyperventilation by the anesthesiologist, and by passing oxygen through the artificial lung only during the period of perfusion. It is of interest that even in those patients in whom pCO_2 was less than 20 mm. Hg, no electroencephalographic abnormalities were observed and all these patients survived. The clinical observations herein reported support experimental evidence that cerebral circulation during perfusion is not significantly affected by the pCO_2 level.[13, 14]

CONCLUSIONS

This clinical study shows that the use of 100 per cent oxygen in an artificial oxygenator does not result in pCO_2 washout. In fact, the pCO_2 remains constant under these conditions and minimal pH changes occur.

On the other hand, addition of carbon dioxide results in a rise in pCO_2 and thereby adds a respiratory acidosis to the metabolic acidosis occurring with perfusion. It is, therefore, concluded that it is not necessary and, in fact, may be detrimental to add carbon dioxide to artificial oxgenators during extracorporeal circulation.

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SIMPLIFIED FLOW DETERMINATION, VOLUME CONTROL, AND BLOOD REPLACEMENT IN THE ROTATING DISC OXYGENATOR

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MECHANICAL DISADVANTAGES in operation of the Kay-Cross oxygenator[1] in combination with the De Bakey roller pumps have been observed by us in clinical and laboratory perfusions. These deficienees include:

1. Rapid changes in oxygenator reservoir fluid level which may be associated with (a) going on bypass, (b) going off bypass, (c) initial removal of blood from within a large heart and the reverse situation when cardiotomy is closed, (d) blood loss in operative field, (e) caval cannula occlusion, and (f) use of a vasopressor.

2. Interference with oxygenator efficiency when fluid level changes within oxygenator. (This can be disastrous if volume change is sudden and large, a situation not infrequent at high flow rates in adults.)

3. Difficulty in rapidly removing or adding blood to oxygenator reservoir in event of fluid fluctuations.

4. Difficulty in accurately determining output of partially occlusive roller pumps.

5. Inability to determine accurately pre-perfusion and post-perfusion volume in oxygenator reservoir because of large cross-sectional area.

6. Occasional formation of small surface bubbles in oxygenator reservoir.

Minor modifications designed by one of us (R.S.C.) have virtually eliminated these problems in our artificial heart-lung: (1) gravity drainage of caval blood to a vertical receiving reservoir of large capacity (2,650 c.c.) but of small cross-sectional area (1 cm. fluctuation = 50 c.c. or with central obturator 1 cm. fluctuation = 25 c.c.) (Fig. 1), and (2) addition of an overflow tap at the desired blood level in the arterial end-plate (Fig. 2).

With these adjuncts a drainage line between the overflow tap and the receiving reservoir bleeds back excess blood, and constant oxygenator reservoir volume is automatically obtained by simply maintaining the output of the venous pump slightly greater than that of the arterial pump (Fig. 3). Prior

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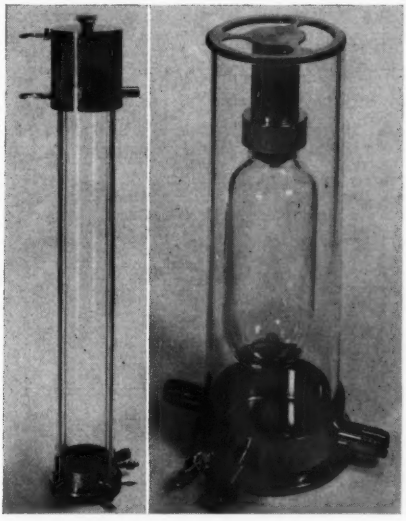

A. *B.*

Fig. 1.—*A*, Gravity drainage receiving reservoir. Pyrex glass cylinder is 51 cm. long with an internal diameter of 8 cm. Taps on base plate are used for (1) caval cannula drainage, (2) venous pump line, (3) overflow line, and (4) left atrial drainage. Intracardiac blood returned through tap on reservoir head is directed to flow down side of reservoir.

B, Central obturator increases accuracy if desired in small patients.

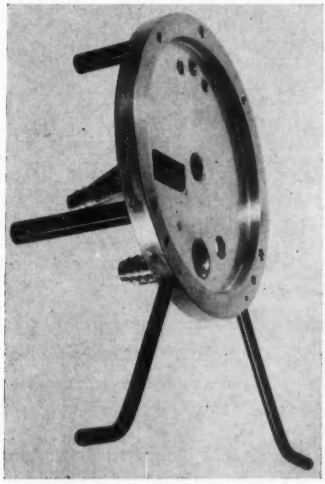

Fig. 2.—Prototype model arterial end-plate with rectangular over-flow tap 4 by 1.5 cm. in size. Lower edge of tap is at height of desired blood level, and inner border is beveled slightly to avoid hold-up due to surface tension.

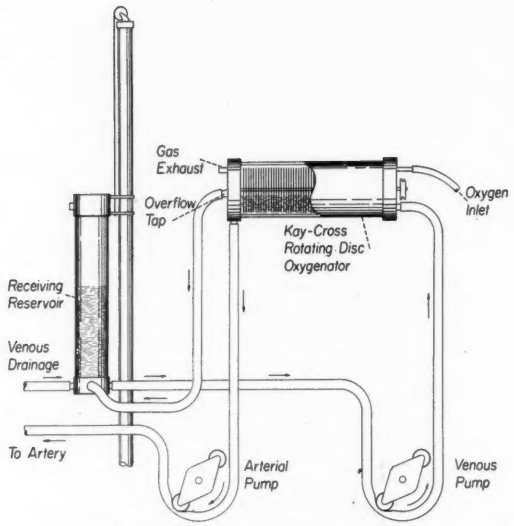

Fig. 3.—Diagram demonstrates flow system used. When venous pump output exceeds arterial pump output, oxygenator reservoir volume is constant.

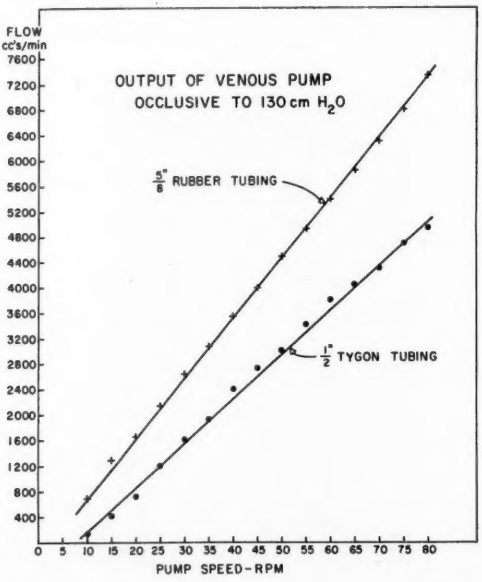

Fig. 4.—Calibrations of venous pump have demonstrated linear relationship between pump speed and output. Rollers are ordinarily set to operate semi-occlusively, resisting back pressure of 130 cm. of water.

to bypass, blood is cycled allowing one to determine variations of the fluid level in the receiving reservoir due to build up on the upturn side of the discs and at the venous end of the oxygenator reservoir. These volumes are usually small but may be considered when regulating extracorporeal volume during bypass. Variation in extracorporeal volume is then reflected in the receiving reservoir where the accuracy of measurement equals that of a graduated cylinder in which 1 cm. equals 50 c.c. volume. In children, the central obturator may be added, a shorter, smaller volume reservoir utilized, and reading accuracy increased with a centimeter fluctuation equaling 25 c.c. (Fig. 1, *B*). An inexpensive pulley system permits control of receiving reservoir height with variation of amount of negative venous line pressure as desired.

Immediate automatic replacement of operative blood loss up to 2,000 c.c. occurs since this blood is carried in the receiving reservoir once bypass is established. Additional blood can be rapidly added from the donor reservoir.

Since input and output resistance is known when pumping from a receiving reservoir to an oxygenator reservoir where volume is constant, the partially occlusive venous pump properly calibrated prior to bypass becomes a reliable flow meter. Fig. 4 shows the linear relationship between pump speed and pump output under these circumstances. Measurement has shown the volume passing through the overflow tube during bypass to be 60 c.c. of blood per minute when a small trickle is maintained. The actual volume can be determined by clamping the overflow line and measuring the time-volume relationship. Naturally, the overflow volume is considered when calculating the arterial pump output on the basis of the venous pump revolutions per minute.

An unexpected advantage is that the overflow tap system efficiently removes the bubbles which form on the surface of the fluid level of the oxygenator where the discs enter the blood.*

Use of this system in bypass procedures has been uniformly satisfactory. An extremely accurate blood balance, avoidance of undesirable fluid fluctuations within the oxygenator reservoir, and immediate replacement of blood loss have materially reduced sources of human error in the operation of this apparatus. The set-up avoids the undesirable rigidity of electrical regulating systems and permits control utilizing the central venous pressure parameter.

SUMMARY

1. Mechanical operation of the Kay-Cross oxygenator with partially occlusive De Bakey roller pumps has been simplified by (a) gravity drainage to a vertical receiving reservoir of small diameter but large capacity, and (b) addition of an overflow tap to the arterial end-plate of the oxygenator reservoir.

2. These modifications have resulted in (a) accurate extracorporeal volume determination, (b) automatic volume control within the oxygenator reservoir, (c) immediate blood replacement of up to 2,000 c.c. in the event of sudden blood loss, (d) reliable flow determination utilizing the partially occlusive venous

*Direction of disc rotation may require reversal in certain commercially available systems since it is desirable to have the lower more stable level facing the pump operator.

pump, (e) removal of surface bubbles by way of the overflow tap, (f) transfer of fluid fluctuations to the noncritical receiving reservoir, (g) avoidance of the undesirable rigidity of electrical regulating systems, and (h) ready control of venous line negative pressure.

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A SYSTEM FOR HYPOTHERMIC PERFUSION

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THE COMBINATION of hypothermia and total body perfusion has many desirable features.[1-7] Rapid changes in the temperature of blood are accomplished by heat exchangers interposed in a heart-lung machine circuit.[6, 7] A new simple technique of internal cooling and warming is described, its safety features discussed, and its advantages stressed.

DESCRIPTION OF APPARATUS FOR INTERNAL COOLING AND WARMING OF BLOOD

Internal cooling is accomplished by pumping water at 4° C. countercurrently through a 325 cm. long, 22-gauge, stainless steel spiral inserted in the Mayon helix of a DeWall-Lillehei heart-lung machine (Fig. 1). The latter is not immersed in the customary water jar, as the heat exchange across the Mayon tubing is minimal.[8] This system is inexpensive, plain in design, easily assembled, and simply cleaned. The blood and water circuits are completely separated and the heat transfer coefficient compares favorably with the Brown exchanger.[7] The priming volume of the helix is reduced without altering the efficiency of the system as a bubble separator.

The internal warming device, in addition, consists of stainless steel tubes bent in the shape of hairpins and placed in the venous reservoir (Fig. 2). Fig. 3 is a diagram of the cooling and warming system used with a DeWall-Lillehei bubble oxygenator for total body perfusion.

TECHNIQUE OF HYPOTHERMIC PERFUSION FOR OPEN HEART SURGERY

External cooling is carried out during the preparatory phase of surgery, and internal cooling is started when cannulation of both venae cavae and femoral artery is completed. The perfusion rate is preset at 20 c.c. per kilogram of body weight and heparin, 1.5 mg. per kilogram of body weight, is given intravenously. At an intraesophageal temperature of 30° C., the aorta is clamped distal to the coronary ostia for periods of 15 to 25 minutes at a time during the repair of the intracardiac defects. Warming is started a few minutes before the anticipated termination of the corrective procedures and the temperature is usually about 36° C. by the time the chest is closed. Polybrene† is usd to counteract heparin.

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†Supplied by Abbott Laboratories, Chicago, Ill.

EXPERIMENTAL DATA

Extensive investigation preceded the experimental and clinical adoption of this technique and will be reported separately. Consistent survival was obtained in a consecutive series of 8 dogs following 30 minutes of aortic occlusion at 20° C. and right ventriculotomy.

CLINICAL DATA

The clinical data on the first 7 consecutive patients who underwent hypothermic perfusion by the technique described are summarized in Tables I-III.

TABLE I. CLINICAL DATA ON 7 PATIENTS WHO UNDERWENT HYPOTHERMIC PERFUSION

PATIENT	AGE (YR.)	WEIGHT (KG.)	DIAGNOSIS	FLOW RATE (C.C./KG. BW/MIN.)	TOTAL BLOOD USED
1	31	70	I.A.D.*	20	3,500
2	43	50	I.A.D.	20	3,000
3	4	16.3	T.O.F.†	20	2,500
4	14	35	T.O.F.	20	7,000 (Blood loss through a patent ductus arteriosus)
5	4	14.5	Pulmonary valvular and infundibular stenoses	20	2,000
6	11	43	I.A.D.; explored for possible pulmonary stenosis	20	2,000
7	35	43	Mitral commissurotomy	20	2,750

*Interauricular defect.
†Tetralogy of Fallot.

TABLE II. HYPOTHERMIC PERFUSION DATA ON 7 CONSECUTIVE PATIENTS

PATIENT	TOTAL PERFUSION TIME (MIN.)	DURATION OF AORTIC OCCLUSION (MIN.)*	DURATION OF CARDIAC REPAIR (MIN.)	TEMPERATURE RANGE DURING AORTIC OCCLUSION
1	38	15†	22	27° C.-30° C.
2	41	13†	20	29° C.-30° C.
3	69	22‡	56	26° C.-30° C.
4	121	66 (20, 23, 7 and 16)‡	110	27° C.-30° C.
5	124	0‡	33 and 30	23.5° C.-31° C.§
6	75	24 (18, 6)‡	41	27° C.-30° C.
7	38	18‡	28	26° C.-30° C.

*Figures between parentheses indicate, in minutes, periods of successive aortic occlusion.
†Ventricles continued to beat regularly.
‡Ventricular fibrillation that responded immediately and reverted to regular rhythm upon allowing coronary flow and applying a single shock of 110 to 135 volts for 0.1 second.
§During cardiac repair.

DISCUSSION

The advantages of this technique for hypothermic perfusion and intracardiac surgery are multiple. The system is clean, inexpensive, locally accessible, and dependable. It decreases the priming volume and preserves all

the features of the DeWall helix. The low flows permitted at these reduced temperatures slow down the "tempo" and allow for lengthy perfusions and rather prolonged periods of repeated aortic occlusions. This offers virtually an empty heart and abolishes the fear of air embolism. The ventricles usually

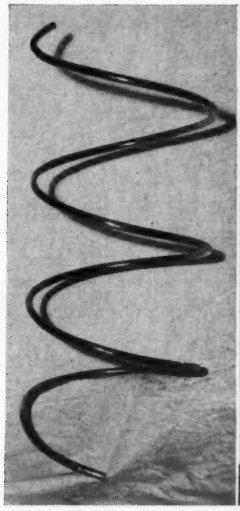

Fig. 1.

Fig. 2.

Fig. 1.—Stainless steel inner helix, a component of the heat exchanger.
Fig. 2.—Stainless steel "hairpins" inserted in the venous reservoir—the second component of the heat exchanger.

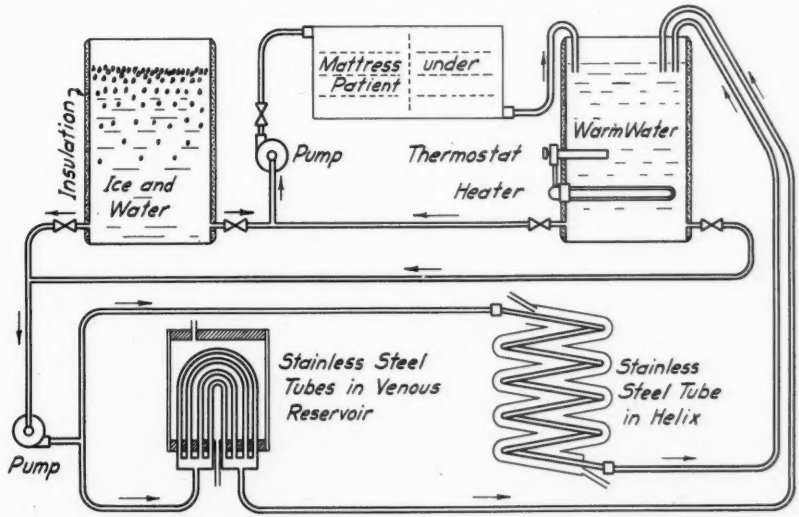

Fig. 3.—A diagram of the entire heat exchange system.

TABLE III. SOME PHYSIOLOGIC DATA ON 7 CONSECUTIVE PATIENTS WHO UNDERWENT HYPOTHERMIC PERFUSION

PATIENT	EEG DURING PERFUSION*	POSTOPERATIVE ECG	RANGE OF PERIPHERAL OXYGEN SATURATION DURING PERFUSION* (PER CENT)	RESPONSE FROM ANESTHETIC	FINAL OUTCOME
1	Adequate	No ischemia or infarction	85-98	Immediate; no pathologic or neurologic signs	Living and well
2	Adequate	No ischemia or infarction	90-97	Immediate; no pathologic or neurologic signs	Living and well
3	Adequate	No ischemia or infarction	90-98	Immediate; no pathologic or neurologic signs	Died on 2nd p.o. day after doing well Autopsy: disruption of I.V. repair
4	Flattening of EEG at beginning due to PDA and then adequate	No ischemia or infarction	85-96	Immediate; no pathologic or neurologic signs	Living and well
5	Adequate	No ischemia or infarction	90-96	Immediate; no pathologic or neurologic signs	Living and well
6	Adequate	No ischemia or infarction	90-96	Immediate; no pathologic or neurologic signs	Living and well
7	Adequate	No ischemia or infarction	—	Immediate; no pathologic or neurologic signs	Living and well

*Traced with Electronics for Medicine Research recorder.

fibrillate after about 15 minutes of anoxia but revert promptly to a regular rhythm upon release of the aorta and applying a single shock of 110 to 135 volts for 0.1 second. Electrocardiograms revealed no signs of ischemia or infarction at the end of the perfusion or postoperatively. Electroencephalograms remained essentially unchanged during the cardiopulmonary bypass. The peripheral oxygen saturations as measured with an earpiece usually read above 90 per cent (see Table III). All patients did well except a patient (No. 3) who died in the second postoperative day after apparently doing well. Autopsy revealed a disruption of the repair of the interventricular defect.

SUMMARY

A simple system for open heart surgery, consisting of hypothermic perfusion and aortic occlusion, is described.

Thanks are due for the unselfish help of Sister Mary Alvera, Modine Pierce, and John Montroy, Jr.

ADDENDUM

Since the submission of this paper, our system was changed to consist of only the inner stainless steel helix for heat exchange. Both the mattress and the hairpin-shaped stainless steel tubes have been eliminated. Up to this date (Feb. 11, 1960), 38 patients had various heart defects repaired with this technique with 30 consecutive survivals. Patient 2 died 4 months after surgery and autopsy revealed an intact interatrial repair and acute pancreatitis. Patient 3 died on the second postoperative day and autopsy revealed disruption of the interventricular repair. Patient 8 died on the third postoperative day and autopsy disclosed intact cardiac repair and severe bilateral pneumonia. Both patients had tetralogy of Fallot.

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OPEN VERSUS CLOSED LEFT ATRIAL DRAINAGE DURING CROSS-CLAMPING OF THE THORACIC AORTA

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THE HAZARDS of cross-clamping the aortic arch distal to the carotid arteries fall into two categories. First is proximal hypertension, which may cause cerebral hemorrhage, cardiac dilatation with ventricular fibrillation, and occasionally pulmonary edema. The second is inadequate blood flow distal to the clamp, which may result in spinal cord damage or ischemic renal insufficiency. The treatment of aneurysms of the distal aortic arch necessitates cross-clamping of the aorta. The method currently employed to obviate these dangers is the pumping of blood from the left atrium to the femoral artery via a closed system.[1] This procedure has not been entirely satisfactory, however, for De Bakey recently stated that 2 of 36 patients so treated have suffered ischemic spinal cord damage following cross-clamping of the descending thoracic aorta, in spite of left atrial to femoral artery bypass.[1] A recent clinical case at the Oklahoma City Veterans Administration Hospital, in which closed left atrial drainage was employed, revealed inadequate proximal decompression with a systolic pressure greater than 250 mm. Hg and transient pulmonary edema in spite of pump flow settings which were increased to greater than 3 L. per minute. This patient developed hypertensive encephalopathy postoperatively, but eventually made a complete recovery. The experience prompted us to study closed left atrial drainage experimentally, and compare it to open, or reservoir, drainage.

MATERIALS AND METHODS

Mongrel dogs, weighing 25 to 35 kilograms, were anesthetized with intravenous Nembutal. Ventilation was maintained by a mechanical respirator. Through a left thoracotomy, a 30 Fr. catheter was inserted into the left atrium. Blood could be drained by either open or closed methods by means of plastic tubing and pumped into the femoral artery via a totally occlusive Sigmamotor finger pump which had been previously calibrated. This pump, when totally occlusive, delivers a constant flow against the pressures encountered in this experiment.[2] A side-arm was provided to measure the actual flows delivered

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(Fig. 1). Pressures were measured in the carotid, femoral, and pulmonary arteries, pulmonary veins, and left atrium by means of polyethylene catheters, Statham strain gauges, and a Sanborn direct-writing oscillograph. Forty-one paired flow measurements of open and closed left atrial drainage were made on 13 dogs, utilizing flow settings between 500 and 2,600 c.c. per minute.

RESULTS

In 40 of these 41 flow measurements, closed drainage delivered less than the calibrated flow (Fig. 2). The single instance in which the flow from closed drainage equaled that of open drainage occurred in a dog which was hypervolemic and at a flow setting of only 500 c.c. per minute.

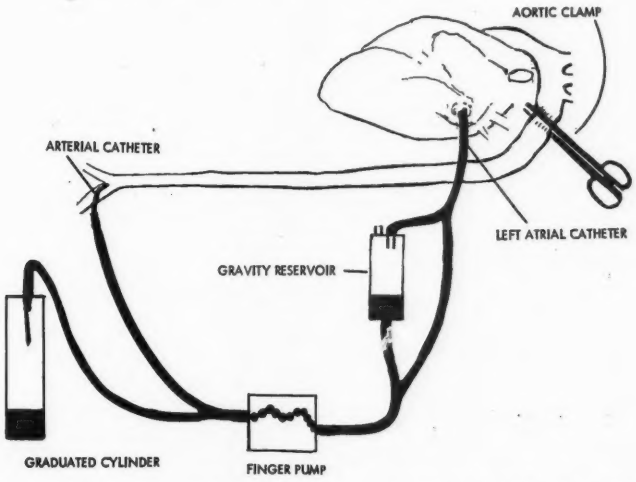

Fig. 1.—Diagram of method for study of open and closed left atrial drainage during left atrial to femoral artery bypass.

The dog's blood volume is an important determinant of the amount of blood drained from the left atrium (Fig. 3). With the pump set at 2,580 c.c. per minute during aortic arch cross-clamping, this volume of blood drained openly from the left atrium into a reservoir and was pumped into the femoral artery. This was the actual volume delivered, for the blood level in the reservoir remained constant. When the system was converted to closed atrial drainage, flow dropped to 525 c.c. per minute. With the measured withdrawal of serial volumes of blood, the amount of flow diminished progressively. After the loss of approximately one sixth of the dog's blood volume, flow had diminished to 195 c.c. per minute at a pump setting of 2,580 c.c. per minute. All 13 dogs showed this progressive decrease in closed bypass flow volumes with serial blood withdrawals; also, with flow settings as low as 500 c.c., progressive decrease in blood volume was accompanied by decrease in flow. Thus, at a setting of 500 c.c. per minute, closed bypass flow as low as 35 c.c. per minute was recorded in

a hypovolemic dog. This relationship is of extreme practical importance, for many surgeons, fearing proximal hypertension during aortic cross-clamping, prefer hypovolemia at this time.

Pressure measurements during thoracic aorta cross-clamping alone are represented in Fig. 4. The pulmonary venous pressure, represented by the mean pulmonary artery wedge tracing, was originally 5 mm. Hg and after aortic cross-clamping increased to 17 mm. Hg. After release of the clamp it rapidly fell to its original level. The arterial side of the pulmonary capillary bed, represented by the pulmonary venous wedge pressure, remained constant at a mean of 27 mm. Hg during this 20-second period of aorta cross-clamping.

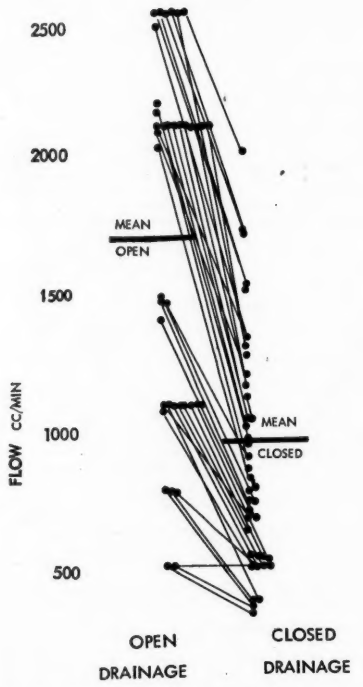

Fig. 2.—Paired flow measurements during open and closed left atrial drainage.

The carotid artery pressure rose to 260 to 280 mm. Hg and the femoral artery pressure fell to 20 mm. Hg during this same interval. During open bypass, left atrial and pulmonary venous pressures were equal and ranged between 10 to 0 mm. Hg. Pulmonary artery pressure was unchanged. Carotid artery and femoral artery pressures were maintained within physiologic limits and varied according to the dog's blood volume and amount of bypass flow. With open left atrial drainage, the reservoir was either raised or lowered until the level remained constant at a given flow setting. If the dog was hypovolemic, left atrial pressure was low and atrial return was diminished. Blood was then

added to the system and in this manner the animals tended to regulate their own blood volume. The same is true clinically of total cardiac bypass in which the caval blood is drained by gravity. Consequently, in these studies, left atrial pressure and bypass flow were regulated by the dog's blood volume. Upon switching to closed drainage, left atrial pressure dropped to –10 to –25 mm. Hg, and occasionally to levels as low as –100 mm. Hg. The pulmonary venous pressure, however, as measured from the wedged pulmonary artery catheter, did not fall, but slowly increased. Pulmonary artery pressure remained constant. The carotid artery pressure rose and the femoral artery pressure fell (Table I). Interestingly enough, if the flow setting was increased at this time, these differences were often magnified.

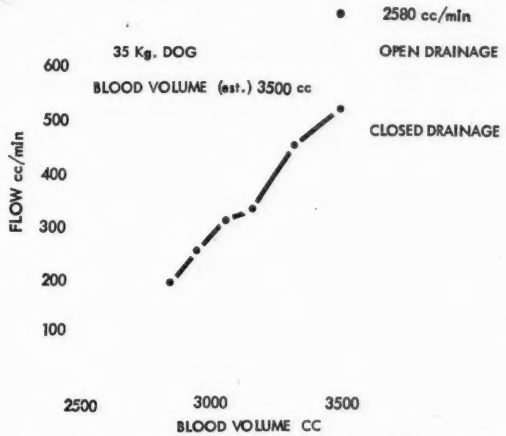

Fig. 3.—Influence of blood volume on bypass blood flow during closed left atrial drainage.

TABLE I. SUCCESSIVE PRESSURE READINGS DURING THORACIC AORTA CROSS-CLAMPING ON CHANGING FROM OPEN TO CLOSED LEFT ATRIAL DRAINAGE DURING LEFT ATRIAL TO FEMORAL ARTERY BYPASS

	OPEN DRAINAGE (2,100 C.C./MIN.)	CLOSED DRAINAGE (520 C.C./MIN.)
Carotid artery	125/110	170/130
Femoral artery	122/96	96/78
Pulmonary artery	18	18
Pulmonary vein	2	16
Left atrium	2	–12

DISCUSSION

Two mechanisms may be involved during closed left atrium to femoral artery bypass to account for the observed pressure changes. The first is collapse of the left atrium due to the negative pressure. This was observed readily in these dogs. This partially occludes the catheter and limits egress of blood from the left atrium. The second is probably the partial or intermittent obstruction of the pulmonary veins at the venous-atrial junction. A valve mechanism

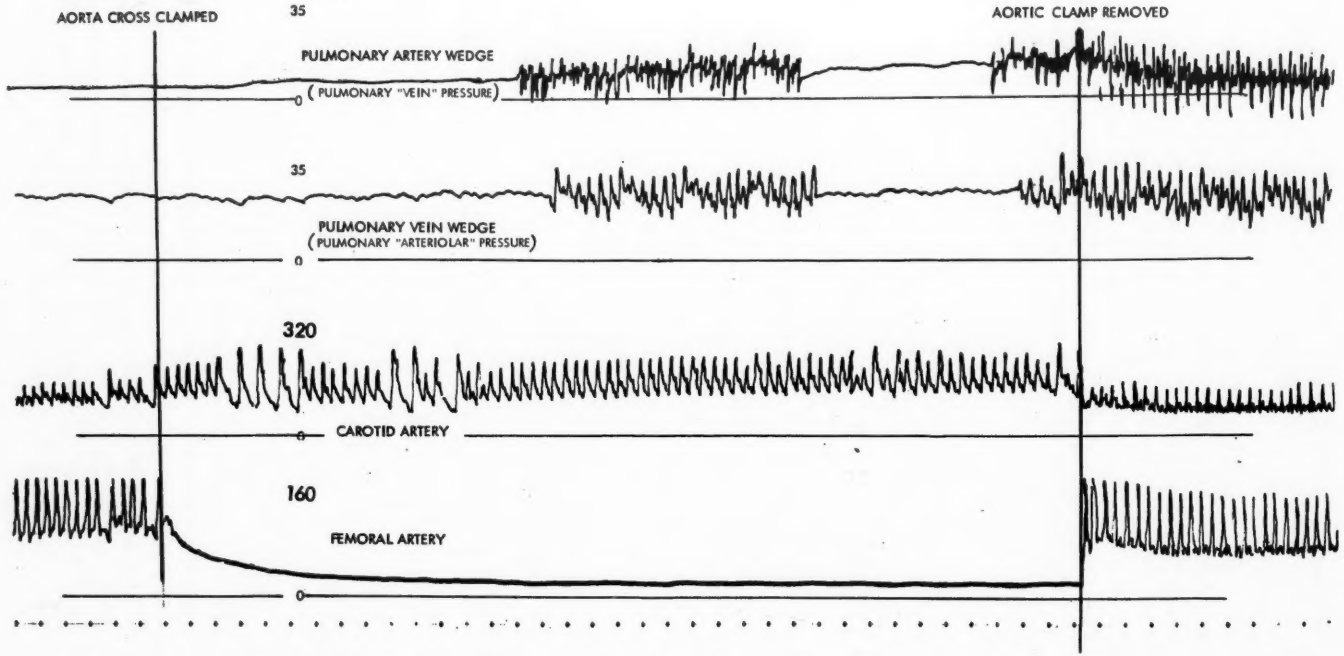

Fig. 4.—Aortic Cross-Clamping Without Vent. Pressure measurements during occlusion of descending aortic arch. (Meaning circuit of oscillograph alternately on and off during pulmonary pressure tracings.)

exists at this junction, for it is known that during normal atrial systole there is very little reflux of blood into the pulmonary veins.[3] This is probably the result of the contraction of atrial muscle around the pulmonary venous orifices. Similarly, with the application of negative pressure within the left atrium, the atrial walls around the vein orifices may be drawn together, partially or intermittently obstructing pulmonary venous return and resulting in an increase in pulmonary venous pressure.

The practical importance of these findings is obvious. Closed atrial drainage may yield inadequate proximal decompression and insufficient flows distally. Increasing the pump flow setting often magnifies these difficulties. The preference for hypovolemia during this period may be disatrous, for it markedly decreases flow. The direct relationship between blood volume and available venous return during extracorporeal circulation has been pointed out by others.[4, 5]

An additional disadvantage of closed drainage which was encountered in these studies was coronary air embolism, invariably fatal in the 5 dogs in which it occurred. This is due to the negative pressure sucking air into the left atrium around the catheter.[6] In the dog, this air lodges in the coronary arteries and can be seen in these vessels.

Open drainage obviates the dangers which result from attempting to suck blood from a closed system. Therefore, during surgical procedures necessitating aortic arch cross-clamping at this institution, closed left atrial drainage has been replaced by open, or reservoir drainage.

SUMMARY

Comparisons were made between open and closed left atrial drainage, during left atrium to femoral artery bypass, in 13 dogs with thoracic aorta cross-clamping. At the same pump flow settings, open drainage yielded greater left atrial drainage with larger and consistently reliable flow volumes to the distal aorta. These differences were magnified in hypovolemic dogs. Mechanisms for differences between open and closed drainage are discussed.

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THE DIFFERENTIATION OF ANEURYSM AND MEDIASTINAL TUMOR

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ONE of the most difficult problems facing the thoracic surgeon is the preoperative differentiation of mediastinal masses presenting in the left chest. These are commonly either carcinoma of the left lung invading the mediastinum, aneurysm of the transverse or descending aorta or, last, one of the various tumors that occur in the anterior mediastinum. Usually the anterior mediastinal tumors can be differentiated preoperatively by their position and configuration on the x-ray films. On the other hand, carcinoma of the lung and aortic aneurysm are often confused, short of thoracotomy. Angiocardiography[1] has provided a means of distinguishing these two latter lesions in most cases. However, its use is not infallible and the following cases are presented to demonstrate some of the problems encountered in lesions in this area and how the various special studies now available are used in making a diagnosis.

CASE REPORTS

CASE 1.—M. L. (S.U.H.—No. 359914), a 54-year-old white man, was admitted to the hospital with a 1-month history of cough, fever, and hoarseness. Chest x-ray studies demonstrated a large mediastinal mass which was interpreted as a possible aneurysm (Fig. 1, *A* and *B*). Left scalene node biopsy and bronchoscopy were negative for tumor, but the latter revealed a paralyzed left vocal cord. The serology was negative. Angiocardiography was performed and demonstrated a sharply defined ascending and descending thoracic aorta (Fig. 2, *B*). In addition, it was noted that the left pulmonary artery did not fill, presumably due to impingement of the mass on the vessel. Thus, a preoperative diagnosis of carcinoma of the lung was made. At exploratory thoracotomy an inoperable squamous cell carcinoma of the lung was found.

Comment: Although a pulmonary neoplasm was considered to be the most likely diagnosis before operation, angiocardiography was helpful in ruling out an aneurysm.

CASE 2.—O. D. (S.U.H.—No. 361467), a 51-year-old white man, entered with a 6-week history of weight loss, malaise, and cough. He had been referred to the hospital after chest roentgenograms had been interpreted as follows: "Examination of the chest reveals a large aneurysm of the aorta which originates from the junction of the transverse portion of the aorta and the descending portion" (Fig. 3, *A* and *B*). Physical examination showed some

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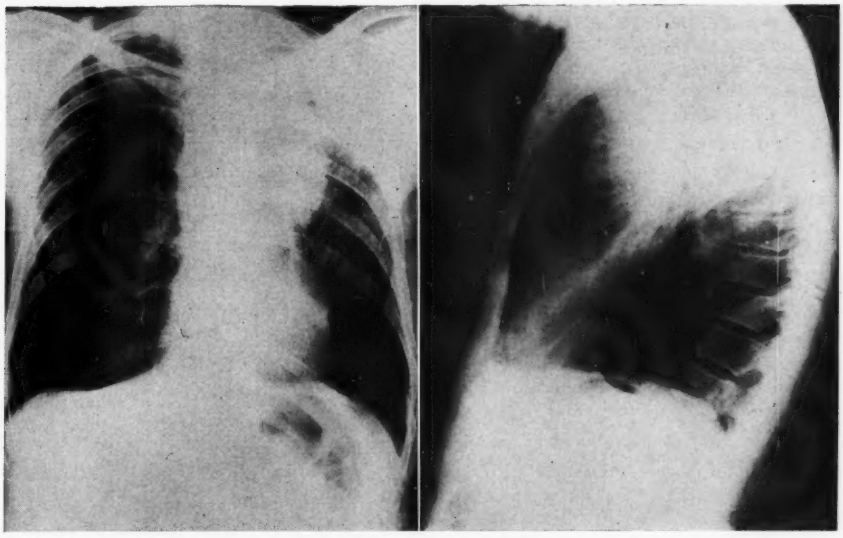
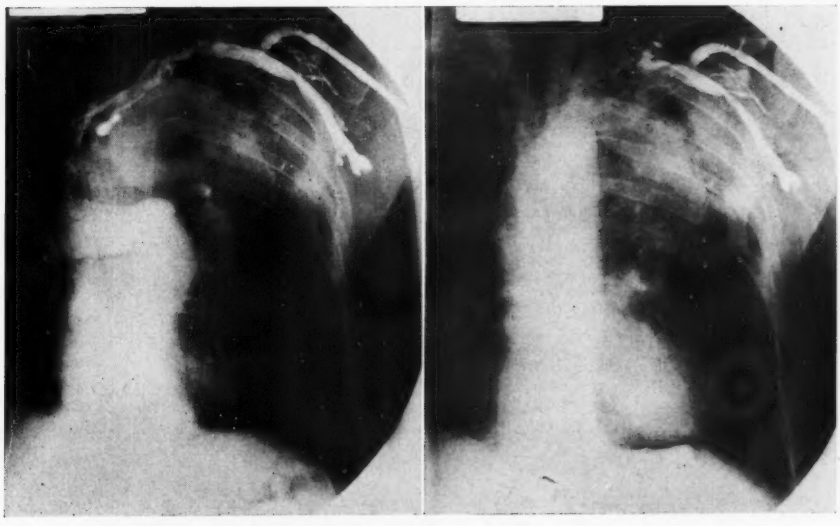


A. *B.*

Fig. 1.—*A*, Posteroanterior chest film of Case 1 which shows a round mass in anterior mediastinum, thought to be an aneurysm. *B*, Lateral chest film of Case 1.

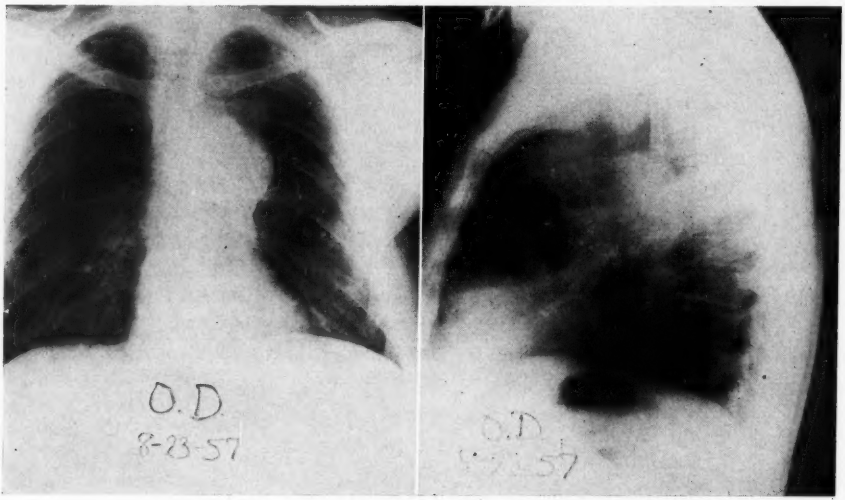


A. *B.*

Fig. 2.—*A*, Angiocardiogram of Case 1 showing absence of filling of left pulmonary artery, presumably due to impingement of the mass on the vessel. The right pulmonary artery is well filled. *B*, Angiocardiogram of Case 1 which shows a sharply defined ascending and descending aorta.

venous distention on the left side of the neck and a palpable left supraclavicular lymph node. The serology was negative. Bronchoscopy and excision of the left supraclavicular node were performed. Anaplastic carcinoma was present in the lymph node. Radiotherapy of the mediastinum resulted initially in shrinkage of the mass.

Comment: This case is of interest since the referring radiologist interpreted the chest roentgenogram as showing an aneurysm. Scalene node biopsy was performed to confirm our clinical diagnosis of carcinoma of the lung. If this had been negative for metastatic tumor, our next procedure would have been angiocardiography.



A. *B.*

Fig. 3.—*A*, Posteroanterior chest film of Case 2 which shows a mediastinal mass interpreted as an aneurysm. *B*, Lateral chest film of Case 2.

CASE 3.—J. M., a 23-year-old white man, had a routine chest film (Fig. 4) taken which demonstrated an anterior mediastinal mass of undetermined etiology. A history of trauma to the left chest 5 years previously was obtained. Because of the possibility of an aortic aneurysm, he was referred to our hospital for further investigation. A retrograde aortogram was performed which was interpreted as showing that the mass was separate from the aorta (Fig. 5). Subsequent exploration revealed a teratoma of the anterior mediastinum.

Comment: Again a diagnosis of aortic aneurysm was strongly suspected on the basis of the chest films. Retrograde aortography correctly demonstrated that the mediastinal mass was separate from the aorta.

CASE 4.—O. H. (S.U.H.—No. 346329), a 59-year-old Negro man, entered the hospital with shortness of breath. He had been treated for syphilis in 1920 and in 1946. On physical examination, he was noted to have a paralysis of the left vocal cord and a systolic murmur in the left fourth interspace. The serology was doubtfully positive. Roentgenograms of the chest (Fig. 6, *A* and *B*) were interpreted as follows, "Huge anterior mediastinal mass with calcific margins. Possibilities are luetic aortic aneurysm, dermoid cyst, or thymoma." After kymograms, the favored radiologic diagnosis was anterior mediastinal cyst, probably dermoid. Since aortic aneurysm could not be excluded, angiocardiography (Fig. 7, *A* and *B*) was

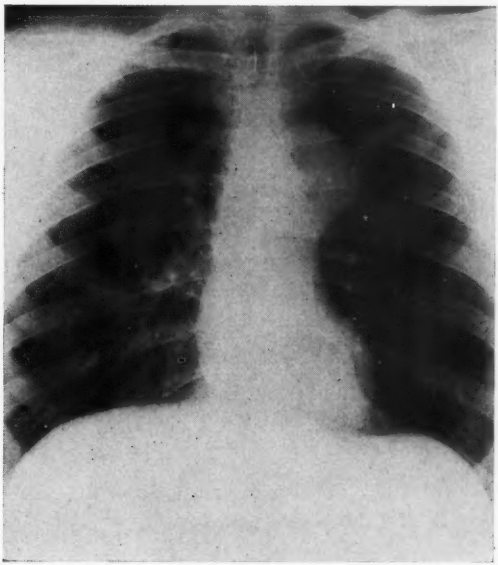
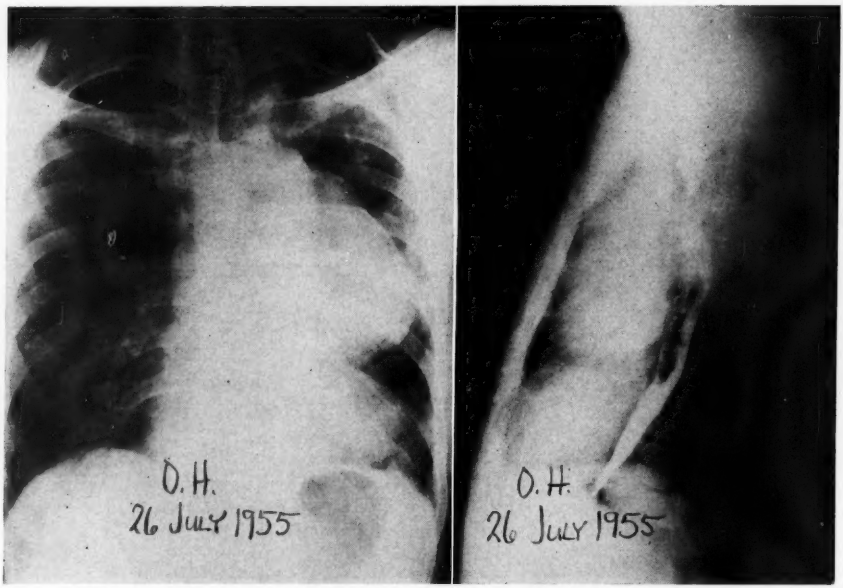


Fig. 4.—Posteroanterior chest film of Case 3 reported as showing a probable aneurysm.

Fig. 5.—Aortogram, lateral projection, of Case 3 interpreted as showing that the mediastinal mass is separate from the aorta.

carried out. The radiologic report on this examination was, "A smooth walled, normally contoured and normal outlined aorta visualized and there is no evidence of any opacification of the large anterior mass. It is felt, therefore, that the latter is definitely not an aneurysm of the aorta. Some widening at the aortic knob is probably due to inferior dipping and bending of the aortic arch." On the basis of this interpretation, the preoperative diagnosis was changed to cyst of the mediastinum. At exploration, a firm rubbery nonpulsating mass was found. This clot-filled aortic aneurysm was inadvertently entered and the patient died (Fig. 7, *C*).

Comment: This case demonstrates that angiocardiographic findings may be misinterpreted when an aneurysm is filled with clot.



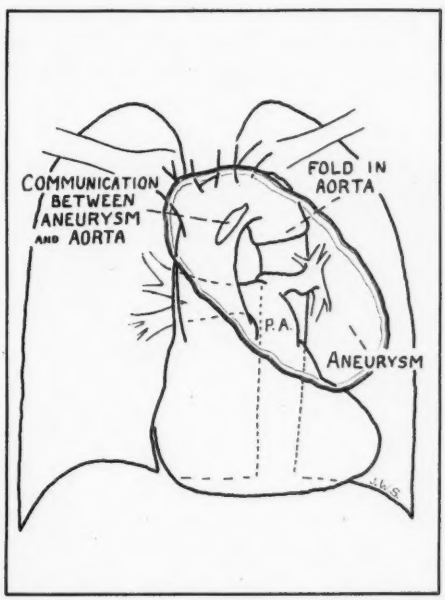
A. *B.*

Fig. 6.—*A*, Posteroanterior chest film of Case 4. The anterior mediastinal mass was interpreted as an aneurysm on the initial films. *B*, Lateral chest film of Case 4 which shows that the mediastinal mass is anterior.

CASE 5.—V. D. (S.U.H.—No. E150098), a 53-year-old white man, sought medical advice because of left chest pain. Chest x-ray studies (Fig. 8, *A* and *B*) showed a left anterior mediastinal mass interpreted as a mediastinal tumor. At exploration, the surgeon found an anterior mediastinal mass that he considered to be an aortic aneurysm. The chest was closed and the patient was admitted to Stanford Hospital, service of Dr. Frank Gerbode, for further care. Review of the chest films again suggested a tumor but angiocardiography seemed definitely indicated in view of the exploratory findings. This study disclosed a sharply defined aortic border and appeared to rule out aortic aneurysm (Fig. 9). At a second thoracotomy, the anterior mediastinal mass was found to be an inoperable carcinoma of the lung, proved by microscopic section.

Comment: This case demonstrates the difficulty in distinguishing mediastinal tumors from aortic aneurysm, even at exploration. Angiocardiography was useful here in arriving at a correct diagnosis.

A. *B.*

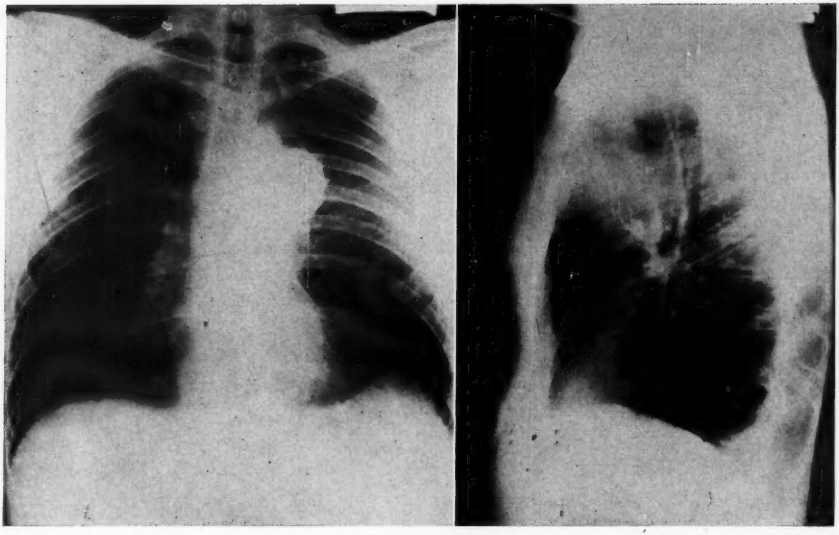


C.

Fig. 7.—*A*, Angiocardiogram of Case 4 which shows poor filling and displacement of the left pulmonary artery.

B, Angiocardiogram of Case 4 interpreted as showing no connection between aorta and mediastinal mass. The tortuosity and folding of the descending aortic arch can be seen. Note the aneurysmal dilatation at the point of the take-off of the innominate artery from the aortic arch.

C, Drawing based on the angiocardiograms of Case 4 and the findings at operation.



A. *B.*

Fig. 8.—*A*, Posteroanterior chest film of Case 5 which shows spherical anterior mediastinal mass with admission diagnosis of an aneurysm of the aorta. *B*, Lateral chest film of Case 5 which shows spherical anterior mediastinal mass.

Fig. 9.—Angiocardiogram of Case 5 interpreted as showing that the anterior mediastinal mass is separate from the sharply defined aorta.

DISCUSSION

A correct preoperative diagnosis in these cases is important because special equipment and abilities must be available if an aortic aneurysm encountered at operation is to be dealt with successfully.

Conventional methods of diagnosis include physical examination, chest x-ray studies, and kymography. Fluoroscopy and kymography may demonstrate the presence or absence of pulsation. Nevertheless, these findings may cause confusion, since solid tumors can transmit the pulsations of the great vessels whereas many aneurysms do not pulsate, due to extensive thrombus formation within. Tomography may be helpful in demonstrating whether or not a mass is continuous with the aortic shadow, but it is not definitive.

With the introduction of angiocardiography and aortography it was hoped that this dilemma would be resolved. Although angiocardiography is usually successful in the differentiation of these masses, this is not always true, and, as seen in Case 4, in spite of what appeared to be an adequate visualization of the aorta, an incorrect radiologic diagnosis was made. Because of this diagnosis, the surgeon was not prepared for the possibility of an aneurysm at the time of thoracotomy.

If the mass in question opacifies by angiocardiography or retrograde aortography, the diagnosis of aneurysm is confirmed. However, a negative report is not so conclusive, as many aneurysms do not opacify. Steinberg and Dotter[2] and Sussman[3] have reported cases in which aneurysms did not opacify with angiocardiography, but a correct diagnosis was made because, in one instance, a second aneurysm was present which did fill with dye, and, in the other, the stigmata of syphilitic aortitis were present. These changes of syphilitic aortitis have been described by Steinberg and his associates.[4] They include aortic dilatation, irregularity of the aortic lumen, calcification of the ascending aorta, aortic tortuosity, and abnormal aortic wall thickness. Many of these findings are more easily demonstrated on left anterior oblique or lateral projections of the angiocardiogram. In Case 4, just reported, we do not know whether the aorta was normal or whether its appearance was misinterpreted as being normal.

Perhaps another view, lateral or oblique, would have demonstrated the syphilitic changes in a clearer manner, and might have resulted in a more successful outcome in this case. This case also demonstrates that an aneurysm of the ascending aorta can present on the left side of the chest instead of its usual projection on the right side. Of the various special techniques now available to differentiate carcinoma of the left lung and aortic aneurysm, visualization of the thoracic aorta either by angiocardiography or aortography is the most reliable.

However, we wish to stress that even with utilization of the best diagnostic tools at hand, the surgeon must be aware of the possibility of encountering either lesion at the time of thoracotomy.

SUMMARY

The preoperative differentiation of aneurysm and mediastinal tumor is important because of the difference in the surgical equipment and abilities necessary to treat these two conditions. The diagnosis can usually, but not invariably, be made by contrast visualization of the aorta. Illustrative cases are presented.

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TUBULAR HYPOPLASIA OF A PERSISTENT RIGHT AORTIC ARCH WITH SURGICAL CORRECTION

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AN UNUSUAL anomaly, consisting of absence of the left aortic arch with tubular hypoplasia of a persistent right aortic arch, was recently encountered. A review of the literature has failed to disclose any similar case report. The method of surgical repair will be described and the embryologic derivation of this anomaly will be discussed.

CASE REPORT

A 16-year-old white male was admitted to The Ohio State University Hospital Feb. 15, 1959, with a diagnosis of coarctation of the aorta. He complained of shortness of breath, chest pain on exertion, and cold feet. The past history indicated that for the first 3 months of life he had frequently choked on his feedings. The blood pressure was 178/90 mm. Hg in the right arm, 110/60 in the left arm, and 100/60 in the legs. A Grade 2 systolic murmur was heard in the left second interspace and in the interscapular area. Femoral pulsations were only faintly palpable.

The electrocardiogram suggested left ventricular hypertrophy. A barium study demonstrated a pressure defect in the posterior wall of the esophagus, suggesting the presence of an anomalous vessel (Fig. 1). The usual left aortic arch indentation of the esophagus was absent as noted in Fig. 2. Instead, the esophagus is displaced to the left.

Operative Findings.—The chest was entered through a left posterolateral incision. The descending aorta was slightly to the right of its normal location. Its proximal end gave rise to a diminutive left subclavian artery (Fig. 3, *D*). There was no evidence of any connection to a left aortic arch. The pericardium was then opened and an enlarged ascending aorta was found. The first branch was the left common carotid, the second was the right common carotid, and the third was the right subclavian artery. At first, it appeared that the ascending aorta ended in these three branches, but further dissection disclosed another branch, approximately the diameter of the carotid vessels (6 to 7 mm.), arising from its posterior wall. This rudimentary right arch passed behind the esophagus and joined the descending aorta 3 cm. distal to the origin of the left subclavian artery. The ascending aorta was markedly pulsatile, whereas the descending aorta pulsated weakly. A ligamentum arteriosum arose from the left pulmonary artery and joined the descending aorta on the anteromedial surface of its saccular termination.

Operative Repair.—A curved vascular clamp was applied to the medial aspect of the ascending aorta parallel to the long axis of the vessel to exclude a portion of its wall.

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Fig. 1.

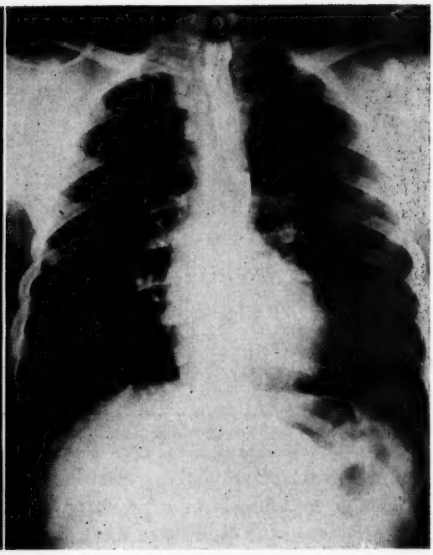

Fig. 2.

Fig. 1.—A left lateral chest roentgenogram demonstrating a pressure defect in the posterior wall of the esophagus caused by the hypoplastic right aortic arch.

Fig. 2.—The esophagogram demonstrates an absence of the usual indentation of the left wall of the esophagus by a left aortic arch. Instead, the esophagus is displaced to the left.

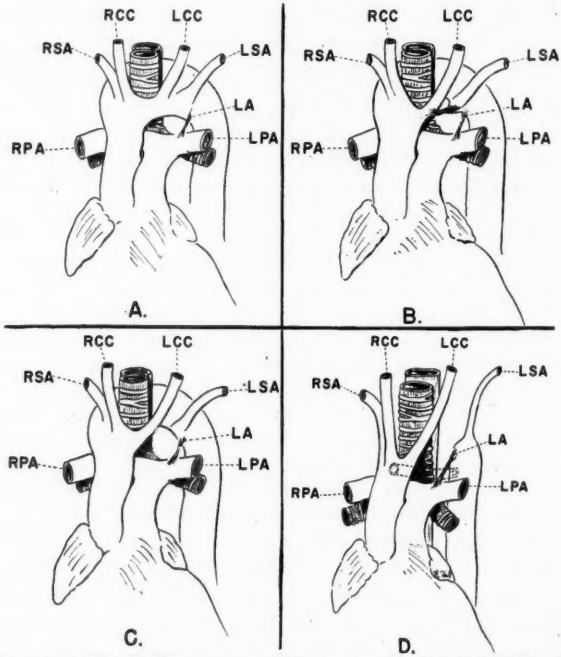

Fig. 3.—These drawings illustrate the successive steps in the derivation of tubular hypoplasia of a persistent right aortic arch. Starting with a functional double aortic arch (*A*), atresia occurs in the left arch between the origins of the left carotid and subclavian arteries. The atretic segment may remain as a fibrotic cord (*B*), or may completely disappear (*C*). Tubular hypoplasia of the right arch has occurred simultaneously (*D*). The left subclavian artery arises from the descending aorta.

Care was taken not to obstruct the flow of blood from the left ventricle. Another clamp was applied in the same way to the descending aorta. A crimped Teflon graft, 5/8 inch in diameter, was anastomosed in end-to-side fashion to a longitudinal incision placed in the excluded portion of each vessel. A continuous 5-0 arterial suture was used (Fig. 4). This resulted in the formation of a vascular ring surrounding the esophagus and trachea, but care was taken to employ a graft of sufficient length to prevent pressure on these structures.

The patient's postoperative course was uncomplicated. Three months after operation the blood pressure in the arms was 150/100 mm. Hg and 148/110 in the legs. A faint systolic murmur remained audible.

Fig. 4.—Operative photograph of the anomaly after completion of the Teflon graft anastomosis.

DISCUSSION

To understand this anomaly, a review of the normal embryology of the aortic arch is helpful.[1] Six pairs of branchial arches appear at different times in embryonic life (Fig. 5). The first, second, and fifth arch on each side disappear. The third aortic arches persist as the common carotid arteries. The right fourth arch forms the proximal portion of the right subclavian artery *(a)* while the segment between the subclavian artery and the dorsal aorta *(b)* disappears. The left fourth arch forms the portion of the adult aortic arch *(c)* between the origin of the left common carotid and the entrance of the ductus arteriosus *(d)*. The ventral part *(e)* of the right sixth arch becomes the right

pulmonary artery and the dorsal part *(f)* disappears. The ventral part of the left sixth arch *(g)* forms the left pulmonary artery, while the dorsal part remains as the ductus arteriosus *(d)*.

The derivation of aortic arch anomalies may be best understood, as suggested by Edwards,[3, 4, 5] by referring to a functional double aortic arch as the basic pattern (see Fig. 3, *A*). Such a functioning double arch does exist at one stage of fetal life.[3]

If atresia of the left fourth arch occurs, it usually involves that portion between the left subclavian artery and the insertion of the ductus arteriosus. However, the atresia may occur between the left carotid and left subclavian

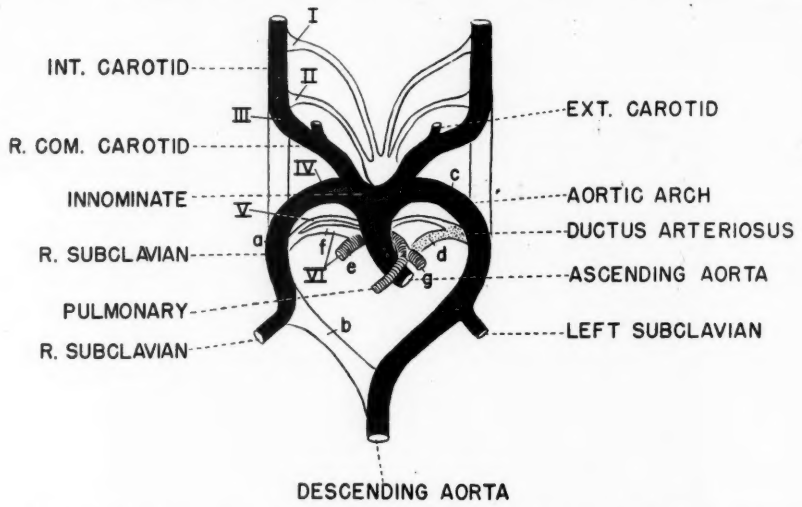

Fig. 5.—A diagram of the six pairs of embryonic aortic arches. The vessels outlined in solid black persist while the vessels in white disappear. The ductus arteriosus (*d*) is stippled to indicate persistence until after birth.

arteries (see Fig. 3, *B*). In this event, the latter vessel arises from the descending thoracic aorta. If the atresia occurs late in fetal life the atretic segment remains as a fibrotic cord. If the atresia occurs at an earlier stage, as in the case presented, no connection between the vessels may be found (see Fig. 3, *C*). The innominate artery depends for its existence on the disappearance of the right fourth arch beyond the origin of the left common carotid artery. If this arch persists, as in our case, the right subclavian and common carotid arteries arise separately (see Fig. 3, *D*).

Edwards has used the term "tubular hypoplasia" to describe cases in which there was a uniform narrowing of a long segment of a left arch. This is to be distinguished anatomically from coarctation with a short segment of stenosis. Both conditions may result in the same functional disturbance. The term "infantile coarctation" has been loosely applied to any narrowing of the aorta proximal to the ductus whether it be a true coarctation, a hypoplastic segment, or an area of atresia. Tubular hypoplasia most frequently involves that portion

of the left arch between the left carotid and the left subclavian arteries. To the best of our knowledge, tubular hypoplasia of the right arch has not previously been recorded.[6] The present case should be properly referred to as one of tubular hypoplasia of a persistent right arch.

Baffes[2] has suggested the use of homologous aortic grafts to correct such defects in which primary resection and anastomosis is impossible. In experimental work on dogs he successfully placed a graft from the side of the ascending aorta to the side of the descending aorta. He found that if the aortic arch was very narrow as in coarctation, or if it were tied off, the graft remained open. However, if the aorta was wide open and the graft was nonobligatory, it tended to shrink and close off within 3 or 4 months. In the present case the hypoplastic right arch was markedly diminished in cross-sectional area and it is expected that the graft will remain open as an obligatory one. It was known to be functioning well 4 months after surgery.

SUMMARY

A previously unreported type of congenital aortic arch anomaly is described. This consisted of an absent left aortic arch and tubular hypoplasia of a persistent right arch. The condition manifested itself clinically as coarctation of the aorta. The embryologic derivation of this anomaly and the method of surgical correction are described.

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PLICATION OF THE AORTIC RING IN THE CORRECTION OF AORTIC INSUFFICIENCY

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IT SHOULD be technically possible to diminish effectively the cross-sectional area of the aortic ring, by approximately one third, by plicating the area of the noncoronary cusp. This conversion of the valve to a bicuspid configuration, without interference with the coronary circulation, might be of benefit in cases of free aortic insufficiency associated with aneurysmal dilatation of the base of the aorta. The validity of these assumptions has been explored by a series of animal experiments, and in one human case.

EXPERIMENTAL OBSERVATIONS

Sixty-two unselected, apparently healthy, mongrel dogs averaging 20 kilograms in weight were utilized in this study. All operations were perfomed under anesthesia maintained by a standard veterinarian solution of Nembutal administered intravenously. Right thoracotomy through the fourth interspace was performed, and respirations were maintained by a pump, rhythmically delivering oxygen under positive pressure through an endotracheal tube. The pericardium was incised vertically, anterior to the right phrenic nerve, and the pericardial flaps were held apart with traction sutures. Umbilical tapes were placed about the superior and inferior venae cavae and ascending aortic arch, respectively, and the azygos vein was ligated in continuity. The right auricular appendage was retracted laterally and inferiorly by means of a traction suture attached to its tip, exposing the base of the aorta in the region of the noncoronary cusp (Fig. 1).

A curved De Bakey clamp was placed on the right anterolateral wall of the ascending aorta and a longitudinal incision, approximately 2 cm. long, was made extending from the base of the aorta distally, in the portion of the aortic wall within the clamp. Inflow occlusion was then produced by tightening the umbilical tapes around the venae cavae, followed by occlusion of the aortic arch. At this point, the De Bakey clamp was removed, and a long curved clamp was inserted through the aortotomy toward the aortic valve. The mouth of the

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clamp engaged one of the valve leaflets and avulsed it. With some experience, it was possible selectively to avulse the noncoronary cusp. Inflow occlusion was then discontinued and, as blood welled up through the aortotomy incision, the De Bakey clamp was replaced, followed by release of aortic arch occlusion.

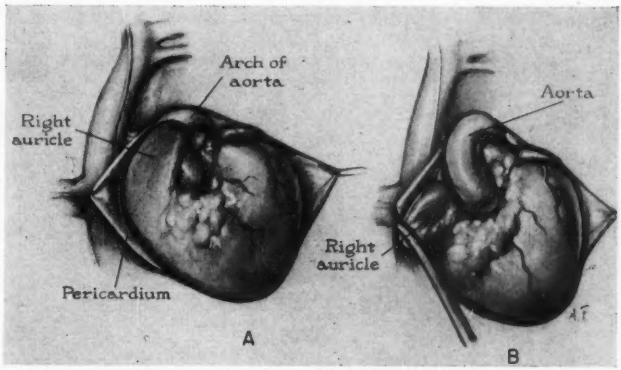


Fig. 1.—*A* and *B*, Exposure of the noncoronary portion of the base of the aorta.

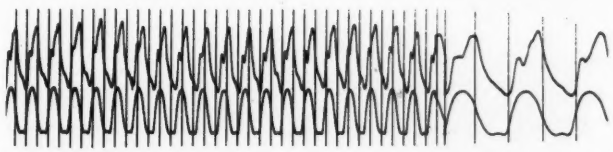


Fig. 2.—Dog No. 59. Pressure curves from aorta (*upper tracing*) and left ventricle (*lower tracing*) following the creation of aortic insufficiency. Note the high amplitude of the aortic curve which equals the left ventricular pressure curve amplitude which indicates marked degree of insufficiency.

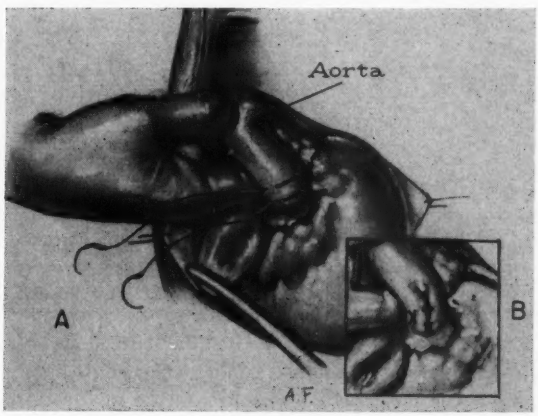


Fig. 3.—Technique for insertion of deep mattress sutures into aortic wall at the base of the aorta over the noncoronary cusp area.

The aortotomy was closed with a continuous over-and-over suture of 5-0 arterial silk. The hemodynamic effect of the valve leaflet avulsion was evaluated by means of simultaneous pressure curves obtained from the left ventricle and ascending aorta and comparing these with similar pressure curves obtained from identical areas prior to creation of the lesion. In the majority of dogs, a definite insufficiency pattern was obtained following creation of the lesion (Fig. 2).

Fig. 4.

Fig. 5.

Fig. 4.—Dog No. 59. Autopsy specimen which shows the sutured aortotomy incision which had been utilized to avulse a leaflet (*upper arrow*), and a pleating mattress suture (*lower arrow*) inserted in an attempt to correct the lesion.

Fig. 5.—Inner aspect of base of aorta following attempted correction pleat.

In the last 15 dogs, an attempt was made to correct the aortic insufficiency immediately following its creation. After the aortotomy wound was closed and the pressure curves were obtained, inflow occlusion of the venae cavae was reinstituted. With the operator's left index finger in the transverse sinus elevating the base of the aorta, one or two large mattress sutures were placed in the aortic wall overlying the noncoronary cusp (Fig. 3). As these sutures were

tied, a large pleat was produced in the aortic wall, in many instances decreasing the circumference of the aortic valve annulus by one third. Figs. 4-6 demonstrate the appearance of a maximal correction pleat, and the sutured aortotomy incision. Following placement of the sutures, inflow occlusion was terminated and, once equilibrium had been established, pressure curves were obtained from the left ventricle and ascending aorta. In spite of the placement of the maximal pleats, the pressure curves repeatedly failed to show any significant change in the insufficiency pattern.

CLINICAL EXPERIENCE

Plication of the aorta in the region of the noncoronary cusp thus proved to be ineffective when applied to the correction of acute aortic insufficiency produced by avulsion of the valve cusp in dogs. On the other hand, a favorable

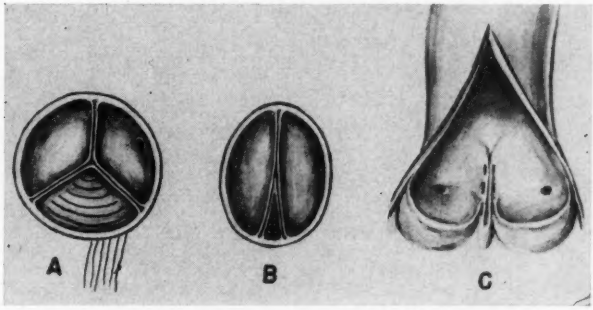


Fig. 6.—Diagrammatic reconstruction of plication of area of noncoronary cusp.

clinical response with a similar method in cases of aortic insufficiency associated with a dilated aortic ring had been reported by Creech[2] with cardiopulmonary bypass and by Hufnagel[6] with a closed technique. The combination of severe angina, free aortic regurgitation, and a hugely dilated aorta comprised the indications for this operation in the following case.

CASE REPORT

G. M., Admission 103408, a 49-year-old Negro male garage attendant, was admitted to the Montefiore Hospital on Jan. 26, 1959, with a chief complaint of angina pectoris of increasing severity, and shortness of breath when lying down. Penicillin had been administered in large dosage shortly prior to hospitalization, when a blood Wassermann test had been found to be positive; he had received no previous treatment, but recalled a penile lesion in his youth. There was no history of rheumatic fever.

Three years prior to admission the patient developed severe pressing anterior chest pain on exertion and severe shortness of breath when lying flat. At this time he consulted a physician who prescribed nitroglycerin tablets, digitalis of unknown dosage, and diuretics which had been used intermittently. The patient experienced relief of the angina with the use of nitroglycerin; however, his consumption progressively increased to as many as one tablet per hour while working. During the course of the past 3 years the patient had had ankle edema on at least one occasion which was relieved by an intramuscular diuretic.

On examination the patient was noted to be a well-developed Negro man who was ambulatory and appeared in no acute distress. The pulse was 88 and regular; blood pressure 180/44 mm. Hg. There was a visible pulsation accompanied by a thrill over the suprasternal notch and bilateral supraclavicular areas. The heart was enlarged to the left, with the point of maximal impulse in the sixth interspace at the mid-clavicular line. All heart sounds were diminished and replaced by loud systolic and diastolic murmurs loudest at the aortic area and transmitted to the apex and right chest. These were accompanied by systolic and diastolic thrills over these same areas, as well as over the carotid, subclavian, and femoral arteries. Liver and spleen were not palpable. An asymptomatic umbilical hernia was noted. There was no ankle edema or clubbing. Neurological examination revealed a slight anisocoria, the right pupil being larger than the left; the right knee jerk was greater than the left.

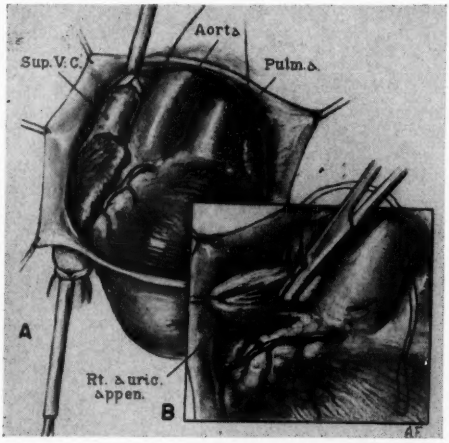


Fig. 7.—Placement of a horizontal mattress suture in one stroke by simultaneous passage of both needles.

Cardiac fluoroscopy disclosed an expansile fusiform aneurysm in the ascending aorta up to the first portion of the arch, with 3+ enlargement of the left ventricle. Electrocardiography showed regular sinus rhythm, with an enlarged left ventricle. The electroencephalogram was normal. The roentgenograms showed marked uncoiling of the aorta, with aneurysmal dilatation of the ascending portion and calcification in the arch. Secondary enlargement of the left atrium and left ventricle was present. The blood serology was positive, with 3+ flocculation and 4+ Kolmer tests. The spinal fluid serology was negative. The venous pressure was 82 mm. of water, with no rise on right upper quadrant pressure. The Decholin circulation time was 18 seconds, arm to tongue.

Plication of the base of the aorta was performed on March 4, 1959. Under satisfactory general intratracheal anesthesia, with a cannula in the left saphenous vein at the ankle, the patient was positioned with the right side of the chest slightly elevated. A right submammary incision was made, and the right pleural cavity entered in the third interspace. There was a generalized diffuse adhesive pleuritis, making it difficult to enter the right pleural cavity. The incision was extended medially, above the left nipple, the left pleural cavity entered in the second interspace, and the sternum was transected with a Lebsche knife. The remnants of thymus were mobilized from the anterior surface of the pericardium, and the pericardium was opened widely, through an incision extending vertically in front of the right phrenic nerve and extended horizontally across the base of the heart. Tapes were passed around the superior vena cava and inferior vena cava, intrapericardially.

The aorta was huge, measuring 5.5 cm. in diameter just above the valve ring, and widening slightly a short distance above this. The right coronary artery was greatly dilated and tortuous. The branches of the left coronary artery were similarly enlarged. The base of the aorta was mobilized on its right lateral and posterolateral aspects, exposing the very base of the noncoronary sinus of Valsalva. With short periods of venous inflow occlusion, none of which extended over 45 seconds in length, a series of 6 horizontal mattress sutures was passed through the noncoronary cusp area, in an attempt to narrow the base of the aorta. The suture material was 1-0 silk, with large curved atraumatic needles on each end of the silk,* passed on a needle holder especially prepared to hold both needles at once, and previously threaded through an Ivalon pillow (Fig. 7). None of the sutures was tied until they had all been placed. During the process of passing these sutures, the heart rate became grossly irregular and finally practically stopped. Intermittent cardiac compression was instituted and the heart went into ventricular fibrillation. While intermittent compression was continued, the sutures at the base of the aorta were all tied anteriorly, over Ivalon pillows. The

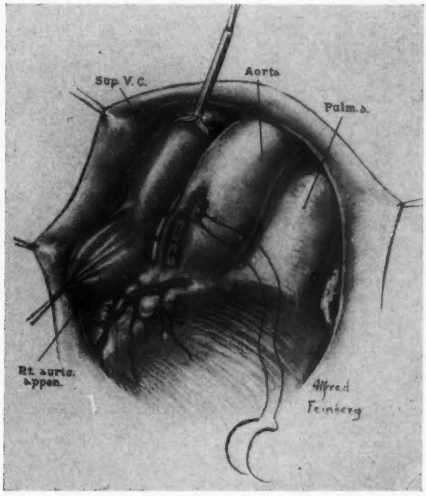


Fig. 8.—Ridge produced by plication of noncoronary cusp area at base of aorta.

heart was then defibrillated with electric shock, two shocks at 50 volts being ineffective, and a third shock at 100 volts arresting the heart, from which it was massaged back into a strong effective beat. Two additional sutures were placed in the plicated area of the aorta, deeply, where a strong thrill could still be felt (Fig. 8).

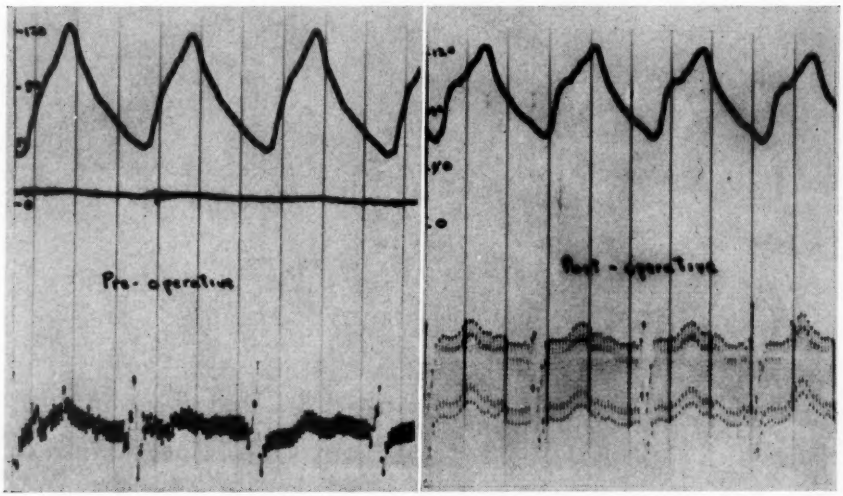
The blood pressure changed from the precorrection level of 160/30 to 130/70 mm Hg. Pulse tracings in the aorta after plication showed a flattening of the dicrotic notch, as compared with preplication (Fig. 9). The plicated base measured 4.4 cm. in diameter, in contrast with 5.5 cm. preoperatively; this effectively reduced the cross-sectional area at this level by approximately one third.

The pericardium was closed loosely. The interspaces were closed with pericostal sutures, with a tube brought out from each pleural cavity in the fifth interspace for underwater drainage. Three sutures of O braided wire were placed to unite the divided ends of the sternum. Closure of the musculature and subcutaneous tissues was accomplished with running chromic sutures, and of the skin with running silk sutures. The patient received 3 units of

*Supplied by the Ethicon Suture Laboratories, Inc., New Brunswick, N. J.

blood during the operation, and his condition at the end of the operation was reasonably good. He reacted completely a short while after operation and showed no evidence of cerebral damage from either his short periods of cardiac inflow occlusion or the period of cardiac resuscitation.

Immediately on return to the recovery room the patient had a blood pressure of 240/100 mm. Hg which gradually came down and stabilized within 5 hours at 130/70. Chest drainage in the first 24 hours was 1,250 c.c. and the patient received 1,500 c.c. of whole blood. On the first postoperative day the blood pressure was 200/? and pulses developed a "pistol shot" character. Symptomatically the patient had no complaints. On the second postoperative day a totally irregular pulse was noted with a rate of 138. The electrocardiogram was interpreted as showing a paroxysmal auricular tachycardia and the patient was started on a rapid digitalization schedule. Venous pressure at this time was 130 mm. of water and circulation time was 22 seconds, as compared to a preoperative venous pressure of 80 and circulation time of 18 seconds. The tachycardia continued with blood pressure maintained at 150/60-40,



A. *B.*

Fig. 9.—Aortic pressure curves before (*A*) and after (*B*) plication operation in case of human aortic insufficiency.

and quinidine sulfate and potassium chloride were added to the therapeutic regimen. On the third postoperative day the electrocardiogram showed an auricular flutter with 3:1 block. On the fourth postoperative day the blood pressure was 160/74-50 with a sharp diastolic change at 74. The patient continued to be comfortable and complained of no anginal attacks.

The tachycardia continued unabated despite use of apparently adequate doses of digoxin, Cedilanid, and Quinidine until March 14. On the ninth postoperative day the ventricular rate slowed to 80, and the electrocardiogram showed atrial flutter. On March 17, 1959, the patient's heartbeat converted to a sinus tachycardia with a ventricular rate of 100, and the course was benign until discharge on April 3. Prior to discharge the blood pressure was essentially the same in both arms, recumbent and sitting, at 140/44 mm. Hg.

When measured 3 months postoperatively the blood pressure was substantially unchanged. The patient could walk 12 to 20 city blocks, in part up hill, without shortness of breath or angina, and had taken no nitroglycerin since the operation.

DISCUSSION

Open heart surgical techniques undoubtedly will provide the optimum solution ultimately of the problems of aortic regurgitation. Bicuspidization of the valve with cardiopulmonary bypass has been reported by Bailey,[1] Garamella,[4] and Hufnagel[6] and their associates for palliation when a dilated aortic ring has been the dominant feature. Efforts at complete valve replacement by a variety of prostheses, when the cusps have been destroyed, have been described with some clinical success by Lillehei[7] and by Hufnagel.[6] Both of these types of open procedures, while clearly indicating the path for the future, must still be considered in the light of the high operative mortality and the short follow-up periods of observation in the survivors. The technique of Gadboys and coworkers[3] for visualization of the aortic valve without occlusion or bypass merits investigation. This widespread concentration of effort reflects the general disinclination to accept the Hufnagel valve, as inserted into the aorta distal to the origin of the left subclavian artery, as an appropriate answer to the problem.

Cases of aortic insufficiency suitable for attempted correction by the plication procedure, while comprising a small percentage of the total, may be selected by appropriate radiologic studies which demonstrate a dilated aortic annulus. The ominous prognosis of this lesion when associated with severe angina pectoris is a strong indication for palliative surgery.

Four technical details in the plication operation, as described here, merit emphasis.

1. Excellent exposure of the noncoronary cusp area at the base of the aorta may be obtained by adequate dissection and mobilization. The boundaries of the cusp itself often stand out clearly, and the origin of the right coronary artery is an additional important landmark anteriorly.

2. Short intermittent periods of venous inflow occlusion permit placement of the plication sutures in a collapsed aorta, with insignificant blood loss.

3. The principle of passing both needles simultaneously for each mattress suture accomplishes two objectives: (a) the 2 component parts of each suture are fixed in a more accurate relationship to each other than would be possible if they were passed separately, and (b) the time for passing each suture (and of each period of venous inflow occlusion) is effectively shortened to about one half of that consumed when each needle is passed separately. The needle holder designed for this purpose has a short copper shelf on one jaw, and is described in detail in a separate communication.[5,*] Passage of these sutures was greatly facilitated by standing on the left side of the operating table, and applying the sutures posteriorly-anteriorly.

4. The hazard of having the sutures erode through the aortic wall may be minimized by tieing them over bolsters on each side of the aorta. Ivalon proved to be completely satisfactory for this purpose. If the bolster intended for the

*Supplied by Codman and Shurtleff, Inc., Boston, Mass.

posterior side of the aorta is placed a short distance back of the 2 needle points, it will help to steady them in the grasp of the needle holder, and will come to rest against the posterior aortic wall when the suture has been passed.

The significance of the relief of angina in the present case, as manifested by discontinuance of nitroglycerin and increased activity by the patient, is difficult to evaluate, particularly in view of the failure to maintain a substantial measurable correction of aortic valvular function as manifested by the wide pulse pressure. Reported relief of angina following a wide variety of procedures emphasizes the unreliability of this criterion. The phase of subjective improvement attendant upon the episode of hospitalization may be properly discounted only after a prolonged period of postoperative observation. The initial reaction to this procedure, however, must be reported as favorable, and may be attributable to increased coronary flow associated with the change in contour of the aortic valve ring.

SUMMARY

1. Plication of the aortic ring has failed to correct aortic insufficiency produced acutely in dogs by avulsion of the noncoronary cusp of the aortic valve.

2. Plication of the noncoronary cusp area at the base of the aorta has been accomplished in a clinical case of free aortic insufficiency associated with dilatation of the aorta, with subjective improvement in the early postoperative course (to date).

3. The principle of simultaneous passage of two needles, swaged onto opposite ends of a strand of silk, permits accurate placement of a horizontal mattress suture in a minimum of time.

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ADDENDUM

April 11, 1960. It is now 13 months postoperatively. The patient has taken no nitroglycerin since operation, and walks stairs without dyspnea. The physical signs of free aortic insufficiency are still present.

MITRAL COMMISSUROTOMY IN PREGNANCY

A Study of 37 Surgical Cases

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PREGNANCY in patients with mitral stenosis always presents to the cardiologist and to the obstetrician many puzzling problems: Will there be any serious risk for the mother during the course of pregnancy? In which conditions is therapeutic abortion indicated? Are there some clinical data on the basis of which it is possible to foresee the behavior of the circulatory system during the following months? Finally, what are the indications and contraindications for a mitral commissurotomy?

Cardiac surgery, greatly developed in the last years, has definitely given the opportunity for a new approach and, often, a solution of these problems. The first results of valvulotomy for mitral stenosis, mostly concerning isolated cases, were reported in the literature in 1952: Cooley and Chapman,[11] Logan and Turner,[22] Brock,[9] Baker, Brock, and Campbell,[8] Rummel,[30] Mason,[24] and, in 1953, A. M. Dogliotti, Actis Dato, and Angelino,[16] Sellors and associates,[32] Oselladore,[27] Burwell and Ramsay,[10] and Parkinson.[28]

The first commissurotomy performed during pregnancy was successfully carried out in 1953 by Prof. A. M. Dogliotti and reported by Dogliotti, Actis Dato, and Angelino. In 1954, larger groups of patients were studied by different authors: Crawshaw and associates[12] (12 cases), Watt[34] (7 cases), Haarston[20] (2 cases), Krauss and co-workers[21] (1 case), Provenzale[29] (2 cases), Mendelson[25] (6 cases), Glover and co-workers[19] (5 cases), Tisne[33] (1 case) and Acevedo[1] (2 cases) and their co-workers, Savi and Mezzasalma[31] (6 cases), Marshall and Pantridge[23] (18 cases), Dellepiane (successful operation during an episode of acute pulmonary edema, and report on 21 operated cases), and O'Connell and Mulcahy[26] (emergency successful operation at the end of the pregnancy).

In the Division of Cardiac Surgery of the Surgical Clinic of our University Hospital we have had the opportunity in these last years of studying thoroughly a fairly large number of patients. It is the purpose of this paper to discuss

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from a surgical, hemodynamic, and therapeutic point of view the indications for mitral commissurotomy in pregnancy and to report on the results obtained in 37 patients.

Hemodynamics of Mitral Stenosis in Pregnancy.—

The pathophysiologic dynamics of the circulation in the pregnant woman with mitral stenosis result from the hemodynamic effects of mitral stenosis along with those induced by pregnancy itself (Actis Dato, Angelino and Levi,[4] Siliquini and Revelli[6]):

a. Oxygen-consumption is increased, more or less remarkably in every case.

b. Arterial saturation of oxygen is not significantly varied.

c. The arteriovenous difference of oxygen is always increased, especially during the second part of gestation.

d. The utilization coefficient of oxygen is increased, owing to the synergic effect of pregnancy-factors (increased coefficient at the uterus-level), and mitral defect (reduced cardiac output at the level of the tissues), and higher oxygen utilization, especially during the second part of pregnancy.

e. Cardiac output and cardiac index are reduced.

f. In pregnancy with mitral stenosis, the systolic output should moderately increase, but, on the other side, the mitral defect causes a reduced output so that finally the output appears generally insufficient.

g. The work of the right ventricle is remarkably increased in relation to the degree of stenosis; the work of the left ventricle depends upon the entity of the defect, but it is generally reduced.

h. Pulmonary resistances are increased in relation to the mitral defect, the peripheral resistances cannot show important variations or appear slightly increased.

From the compiled data, the conflict between the cardio-circulatory troubles, due to the mitral defect and the necessity of increased work caused by pregnancy, appears rather evident. This is the physiopathologic basis for the considerable deterioration of the circulatory condition which is brought about by pregnancy in patients with mitral stenosis.

MATERIAL

The present study was carried out on a group of 37 women. The ages were between 21 and 43 years; within these limits the patients were very irregularly distributed; however, the approximate mean age was 29 years.

History.—

A definite history of rheumatic fever was present in 9 subjects; in 15, repeated episodes of acute tonsillitis were reported; in 5, there was no evidence of previous rheumatic disease. Peripheral emboli had occurred in 10 out of 37 patients (27 per cent), prior to the time of surgery. Twenty-two patients had had 1 to 4 pregnancies in the past; 6 had had spontaneous abortions; 11 were at their first pregnancy at the time of this study.

The general symptomatology of circulatory insufficiency, such as, dyspnea, easy fatigability, palpitations, episodes of pulmonary edema and/or hemoptysis, etc., had become more evident with the beginning of pregnancy and became worse in the following months.

Clinical and Laboratory Findings.—

Auscultation: In 19 cases there was the auscultatory and phonocardiographic pattern of pure mitral stenosis—diastolic rumble, opening snap of the mitral valve, loud and sometimes split second sound. In addition to these signs, a systolic murmur was present in 5 cases over the pulmonary area, in 4

TABLE 1 - RHEUMATIC HISTORY IN OUR PATIENTS

TYPICAL RHEUMATIC INFECTION	17 CASES
TONSILLITIS	18 CASES
NO RHEUMATIC HISTORY	5 CASES

TABLE 2 - EMBOLIC EPISODES BEFORE OPERATION

BRAIN EMBOLISMS	6 CASES
KIDNEY EMBOLISMS	1 CASE
LIMBS EMBOLISMS	3 CASES

TABLE 3 - PREGNANCIES BEFORE OPERATION

NO PREGNANCIES	IN 11 CASES
FROM1 TO 4 PREGNANCIES	IN 20 CASES
ABORTION (1 OR MORE)	IN 6 CASES

over the aorta, and in 6 over the mid-precordium. On the basis of their aspect and comparison with the other clinical data, these murmurs were considered to be of a functional nature. In 3 cases, a short systolic apical murmur radiating to the axilla suggested the association of a moderate mitral insufficiency.

Electrocardiogram: Thirty-three patients had normal sinus rhythm, 4 had auricular fibrillation; 30 out of 37 showed definite evidence of right ventricular hypertrophy.

Roentgenologic findings: Standard x-ray examination (tele-cardiogram in the posteroanterior position, right and left oblique views) showed, in most cases, a typical mitral configuration, with pulmonary congestion (see Tables I-IX).

Surgical Procedure.—

All patients have been operated upon between the third and the seventh month of pregnancy, more frequently during the third or the fourth month.

Operative findings: Thirty-two patients had pure mitral stenosis, in 4 there was some degree of mitral insufficiency, in 1 a moderate aortic insufficiency.

The surface of the mitral orifice was between 0.3 and 1.0 cm.^{2} in 31, and between 1.1 and 1.5 cm.^{2} in the other 6.

TABLE 4 - AUSCULTATION AND PHONOCARDIOGRAM

CHARACTERISTIC FINDING OF PURE MITRAL STENOSIS	19 CASES
FINDING OF PURE M.S. + SYSTOLIC MURMUR IN PULMONIC AREA	5 CASES
FINDING OF PURE M.S. + SYSTOLIC MURMUR ON THE AORTA	4 CASES
FINDING OF PURE M.S. + SYSTOLIC MURMUR IN MESOCARDIAC AREA	6 CASES
FINDING OF DOUBLE MITRAL DEFECT	3 CASES

TABLE 5 - ELECTROCARDIOGRAPHIC FINDINGS

SINUS RHYTHM		32 CASES
AURICULAR FIBRILLATION		4 CASES
ELECTRIC AXIS	TO RIGHT	29 CASES
	IN MIDDLE POSITION	8 CASES
RIGHT VENTRICULAR HYPERTROPHY		30 CASES

TABLE 6 - ROENTGENOLOGICAL FINDING

STASIS OF PULMONARY VESSELS	(++++)	3 CASES
	(+++)	16 CASES
	(++)	15 CASES
	(+)	3 CASES
ENLARGEMENT OF LEFT ATRIUM	(++++)	4 CASES
	(+++)	16 CASES
	(++)	14 CASES
	(+)	3 CASES
ENLARGEMENT OF PULMONARY ARTERY	(++++)	4 CASES
	(+++)	15 CASES
	(++)	10 CASES
	(+)	8 CASES
HYPERTROPHY OF LEFT VENTRICLE		14 CASES

The edges of the valve were elastic in 25 cases, sclerotic in 9, and widely calcified in 3. In 3, there were auricular thrombi. After the operation the mitral orifice was between 2.1 and 2.5 cm.^{2} in 4 women, between 2.6 and 3.0 in 15, between 3.1 and 3.5 in 10, and between 3.6 and 4.0 in 8.

Follow-up study: Frequent studies at regular intervals have been made for 1 to 5 years after operation.

TABLE 7 - GESTATION PERIOD AT TIME OF OPERATION

THIRD MONTH	12 CASES
FOURTH MONTH	18 CASES
FIFTH MONTH	4 CASES
SIXTH MONTH	2 CASES
SEVENTH MONTH	1 CASE

TABLE 8 - INTRAOPERATIVE FINDINGS

PURE MITRAL STENOSIS	32 CASES
MITRAL STENOSIS WITH MITRAL INCOMPETENCE	4 CASES
MITRAL STENOSIS WITH AORTIC VALVULAR DISEASE	1 CASE

CONDITIONS OF THE VALVES

ELASTIC TYPE	25 CASES
SCLEROTIC TYPE	9 CASES
CALCIFIED TYPE	3 CASES

PREOPERATIVE SIZE OF MITRAL OSTIUMS

CM.^{2}	NUMBER OF CASES
0.3-0.4	8
0.5-1	23
1.1-1.5	6

POSTOPERATIVE SIZE OF MITRAL OSTIUMS

CM.^{2}	NUMBER OF CASES
2-2.5	4
2.6-3	15
3.1-3.5	10
3.6-4	8

TABLE 9 -

CARDIO-VASCULAR CONDITIONS AFTER OPERATIONS AND DELIVERY

GOOD OR SATISFACTORY CONDITIONS	32 CASES
LITTLE IMPROVEMENT	5 CASES

RESULTS

Maternal Mortality.—No deaths, either during the operation or postoperatively, occurred in this series.

The cardiocirculatory conditions after operations were very markedly or satisfactorily improved in 32 women; in 5, however, a general improvement but a slight exertional dyspnea and palpitation were still present. One of these had an associated aortic insufficiency and the other 4 an associated mitral insufficiency, clinically diagnosed before the operation.

Course of pregnancy and delivery: Thirty of our patients had a normal delivery, 16 of them with forceps, with no complication of cardiac or circulatory insufficiency. Three women recently operated upon were still pregnant at the time this report was written; they are having a normal gestation without cardiac discomfort. In 3, intrauterine death of the fetus followed the operation; 1 patient had a successful cesarean section at the end of a normal gestation.

DISCUSSION AND CONCLUSION

Clinical Observations.—

The embolic episodes before operation have been extremely frequent in this series of patients; the incidence of 27 per cent being higher than that usually found in mitral stenosis (Actis Dato and Weisz,[5] an analysis of 1,074 consecutive cases). This difference is not easily explained since most of our patients were of comparatively younger age and in sinus rhythm. As far as pregnancy is concerned, however, no definite relation appears to be because all reported embolic episodes had occurred before pregnancy. Another interesting observation was the fact that 26 of our 37 patients had had in the past one or more pregnancies, most frequently followed by a normal delivery; however, the last pregnancy was accompanied by a more or less marked aggravation of their clinical symptoms. In fact, deterioration of the clinical condition of the patients, with increasing palpitation, dyspnea, frequency and severity of attacks of pulmonary edema or hemoptysis, was noted at the beginning of the last pregnancy.

We can state that most of the women subjected to mitral commissurotomy in our Centre came to the hospital not only on the advice of the physician but mainly because of the severe impairment of their cardiac status, which they had not experienced in the previous pregnancies. Other patients whose previous pregnancies had to be interrupted came to the cardiac surgeon with the hope of being cured and becoming able to carry on normal gestation and delivery.

The causes responsible for the aggravation of the clinical symptoms of mitral stenosis during pregnancy are several and complex. The increased load on the heart imposed by the changed metabolic conditions, oxygen consumption, and cardiac output plays evidently the most important role. However, another important factor is probably the increased water retention, due to hormonal influences, which can further aggravate the phenomena of stasis and congestion. We wish to emphasize finally that in most of our cases there was a very tight mitral orifice; it is reasonable to assume that in these subjects with cardiac conditions already impaired that the load imposed by gestation will cause most severe signs of insufficiency.

In 14 cases there was a systolic murmur but no valvular defects could be demonstrated by hemodynamic or clinical data, nor by observations at operation. In fact, most of the murmurs disappeared or were greatly reduced after commissurotomy or after delivery. They are very likely due to the decreased circulation time and do not pathogenetically differ from those in many other physiopathologic conditions, such as hyperthyroidism or anemia. We wish to emphasize the importance of these murmurs, which are very frequent in the pregnant women. A correct recognition of their functional (or inorganic) nature is necessary in order to obtain an exact diagnosis.

The majority of our patients (32 out of 37) were in sinus rhythm; the percentage of auricular fibrillation (about 12 per cent) is definitely much lower than those in the general population who have mitral stenosis who undergo surgical treatment. The reason for this discrepancy is probably that all our patients were of a comparatively younger age and did not show important myocardial damage.

In all of 32 subjects with pure mitral stenosis, x-ray examination had shown some degree of left ventricular enlargement. This phenomenon is probably related to the circulatory changes which take place during gestation, since cadiac rate, at least at rest, does not show considerable modifications. An increase in cardiac output can only be accomplished by an increase of stroke volume, hence of the size of the heart.

In the study of our patients we have paid considerable attention to the problems of the choice of the safest time for operation: 30 patients were operated upon in the third and fourth months of pregnancy, while in the other 7, operations were done in the fifth, sixth, and seventh months.

Some cases have been reported in the literature (O'Connell,[26] Marshall and Pantridge[23]) in which a successful commissurotomy was performed as late as the eighth and ninth month. In our own experience, however, the best period for operation is the fourth month and, actually, it should be performed as soon as possible.

In no case was there a complication of postoperative recurrence of rheumatic fever. According to some authors the endocrine changes occurring during pregnancy act favorably in preventing rheumatic manifestations.

Our patients who had been operated upon were kept under careful medical control and continued digitalis treatment. After a complete cardiological and obstetrical follow-up study through gestation, delivery, and puerperium, we may state that pregnancy by itself does not increase in any way the risks, the difficulties, or the complications of mitral commissurotomy.

Indications for Mitral Commissurotomy During Pregnancy.—

On the basis of our data it is possible to establish some criteria which will help the cardiologist and the obstetrician to solve this important problem, which they always have to face.

1. The knowledge of the cardiac status during previous pregnancies is an indirect datum of great help in the prognosis of the disease during the present gestation; a history of previous pregnancies without any sign of cardiac or

circulatory failure may orientate the physician toward medical rather than surgical treatment, which is, on the contrary, indicated if cardiac abnormalities develop.

2. A progressive deterioration of cardiac conditions before and/or during gestation is a definite indication for prompt surgical treatment.

3. The sudden occurrence of an attack of pulmonary edema is usually a symptom of the downhill tendency of hemodynamic conditions and is therefore a clear-cut indication. In one of our patients an emergency operation was performed at the third month during an acute attack of pulmonary edema which promptly ceased at the moment the mitral orifice was dilated.

4. In cases of combined valvular lesions (associated mitral and aortic lesions associated stenosis, and insufficiency of the mitral valve), the problem of surgical treatment is always very difficult even in the absence of a pregnancy, and needs a careful consideration of every single situation, possibly before pregnancy begins. If an aortic stenosis is associated with mitral stenosis, a combined mitral and aortic valvotomy can be advised and successfully perfromed. If, on the contrary, a severe aortic or mitral insufficiency is associated, the chances of successful medical treatment of an already failing heart are very small. It is also extremely difficult to evaluate the possibilities of continuing a normal gestation, and of accomplishing a normal delivery without danger to the mother. The prognosis of these patients will certainly improve when a reliable method of surgical repair of the combined lesions is possible.

5. We think it is a good policy to perform the operation even in the mild case. The aim of cardiac surgery must be, in fact, not only to cure the established symptoms, but also to prevent the occurrence of future manifestations, which can appear in the last half of pregnancy, particularly during the delivery.

It can conclusively be stated that commissurotomy must be advised and performed without irrational doubts or fears in every case of pure mitral stenosis, the best time being the first months of gestation.

Mitral stenosis is not an indication for a therapeutic abortion.

SUMMARY

Clinical and instrumental findings in 37 women operated upon by mitral commissurotomy during pregnancy are reported.

The maternal mortality was zero, and immediate and long-term clinical results were satisfactory. Three postoperative abortions occurred at various intervals after the operation.

The hemodynamic conditions in pregnant women with mitral stenosis are described in detail on the basis of the writers' personal observations.

The criteria for the evaluation of the indications for mitral commissurotomy during pregnancy are discussed.

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EXPERIMENTAL DELAYED OCCLUSION OF THE LEFT CORONARY ARTERY

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SURGICAL THERAPY for coronary artery disease has met with two imposing problems. On the experimental side, there has been confusion and conflicting evidence on the type of experimental occlusion and consequently there has been inconclusive assessment of the value of an experimental "therapeutic" procedure. On the clinical side, the disease is a potpourri, and case selection, as well as evaluation of therapy, has revolved around the symptom of angina pectoris (the number of attacks, the quantity of nitroglycerin used, etc.), or the presence of historical and electrocardiographic evidence of prior myocardial infarction. Assessment of the patient with angina is enigmatic and judgment of the prognosis of the individual patient in the second group, for purposes of evaluation of any therapy, has proved difficult and requires a large number of cases as seen in the decoagulant studies of recent years.[1]

We feel that the group of patients undergoing acute critical infarction and not responding to medical therapy, who usually die, would constitute an evaluable set of patients if treated and preserved by surgery (almost certainly of the direct type). It might be noted that the effect of the direct surgical attack in the chronic case is presumptive and has not been properly tested in the experimental animal.

The appropriate test animal would be one undergoing a coronary occlusion that is fatal within a few days, and preferably occurring in the awake, closed-chest subject.

Acute ligation of the anterior descending branch of the left coronary artery has been used as a standard and is said to result in a 70 per cent mortality in dogs.[2] However, studies in various laboratories have shown wide variance in results, with mortality ranging from 0 to 100 per cent.[3] The differences are apparently due to many factors: site of ligation, inclusion of the main septal branch, anatomic variations, antecedent medication (especially atropine), anesthesia, etc.[4] In order to minimize some of the variables, slow occlusion in an

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unanesthetized animal with a closed chest has been introduced and used more widely. This preparation should give more consistent results in addition to more nearly simulating coronary artery disease in man. Many methods have been employed to produce a slow occlusion: multiple acute ligations[5]; application of silver clips[3, 6]; encompassing the artery with periosteum,[6] cellophane,[7] bakelite rings,[7] silver bands[3, 8]; spring clamps with jaws separated by catgut[9]; screw clamps left inside the chest[10, 11]; screw clamps[12, 13]; or snares[14] with a controlling device implanted under the skin; radioactive material[15]; injecting sclerosing or fibroplastic agents[16]; and cannulating with a small polyethylene tube.[17] Most of these slow occlusions have been applied to one of three coronary arteries: right, circumflex left, or anterior descending left.

This laboratory has been interested in the direct surgical approach to the problem of coronary artery disease rather than the introduction of multiple small new channels.[18] Slow occlusion of the main-stem left coronary artery seemed to offer a major site of occlusion, segmental in nature, and a coronary artery of adequate size for application of some form of direct surgical approach would remain. The length of the main-stem left coronary artery, before it divides into the anterior descending and circumflex rami, is less than 1 mm. in some dogs and as long as 4 mm. in others. The method of occlusion must be tailored to these anatomic limits. The purpose of this paper is to describe the method evolved and the subsequent results.

In 1956, Berman[19] introduced Ameroid, a casein plastic which has the property of absorbing water when bathed in a salt solution and thus slowly expanding in tissue. He produced gradual vascular occlusion in a number of vessels including the coronary arteries. Litvak, Siderides, and Vineberg[20] made sleeves of Ameroid and placed them around the anterior descending and circumflex branches of the left coronary. Since we were interested in occluding the main-stem artery, the use of Ameroid in sleeves was impossible.

METHOD

Young adult, healthy, mongrel dogs, weighing 11.6 to 21.8 kilograms, were anesthetized with intravenous veterinary pentobarbital sodium and intubated with a cuffed endotracheal tube. Respiration was controlled by manual compression of an inflated bag which delivered 100 per cent oxygen in a closed circuit McKesson system containing a carbon dioxide absorbent. With the right side down, the left hemithorax was entered through the fourth or fifth intercostal space. The pericardium was incised anterior and parallel to the phrenic nerve. A traction suture was placed in the left auricular appendage and the pulmonary artery was gently displaced anteriorly. The epicardium and fat were incised to expose the main stem of the left coronary artery. Number 4-0 silk on an atraumatic needle was passed around the main stem by passing the blunt end of the needle first so as to avoid piercing the artery or its septal branch. Care was taken to pass the suture as close to the aorta as possible. This suture was tied to a No. 0 black silk ligature which was then pulled through (Fig. 1). A wafer of Ameroid, 5 mm. in length, 6 mm. in diameter,

and grooved on two sides, was then tied down over the main-stem left coronary artery (Fig. 2). The pericardium and chest were closed in routine fashion; tube suction drainage of the pleural cavity was performed for 4 to 6 hours postoperatively to insure full re-expansion of the lungs. Broad spectrum antibiotics, usually tetracyclines, were given daily postoperatively.

Fig. 1.

Fig. 2.

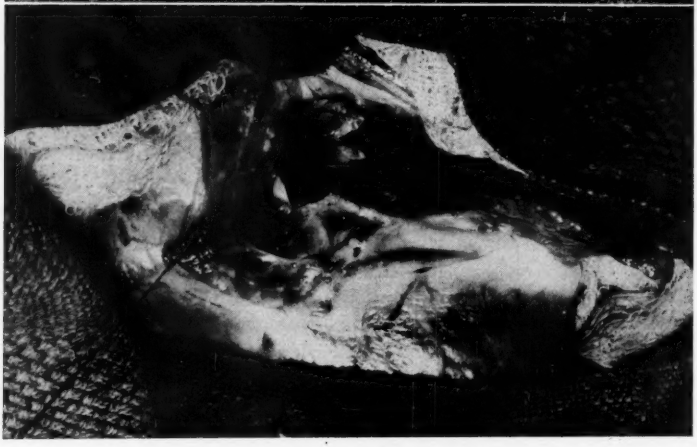

Fig. 1.—The left coronary artery with the ligature in place as seen at operation. The left auricular appendage is retracted to the right. The pulmonary artery is seen above and to the left of the main-stem artery.

Fig. 2.—The Ameroid wafer is tied in place over the main-stem left coronary artery.

RESULTS

Twenty-nine dogs were prepared by the method described. Twenty dogs died between 4 hours and 10 days postoperatively and all exhibited severe occlusion of the main-stem left coronary artery, except 1 which died of pneumonia (Table I). The remaining 9 dogs were sacrificed in 1 month and showed widely

patent or minimally occluded main-stem arteries; the Ameroid and ligature were in place, but had not been tied down adequately.

Electrocardiograms were obtained preoperatively, immediately postoperatively, and almost daily thereafter. All tracings were obtained with the dogs in the supine position in order to minimize variations due to positional changes. No animal showed changes diagnostic of myocardial infarction. Transient S-T changes were common. Arrhythmias were common, ranging from ventricular extrasystoles and left bundle branch block to ventricular fibrillation. Terminal ventricular fibrillation was demonstrated in 4 dogs (700, 88, 618, and 152).

The animals were autopsied as soon after death as possible. The diameter of the Ameroid wafer was measured. Approximately one-half of the swelling occurred in the first 24 hours; i.e., 6.0 mm. to 6.7 mm. or an increase of 0.7 mm. (Fig. 3). Because the occluding ligature encircles the wafer, the circumfer-

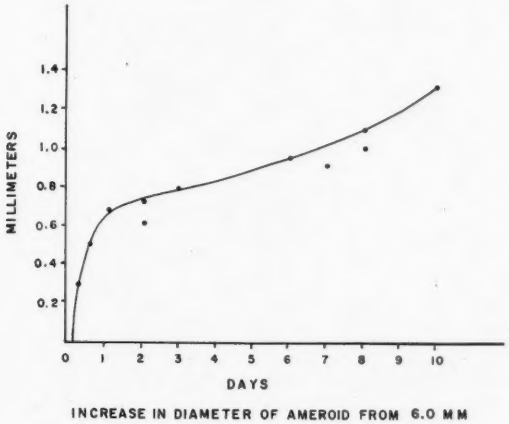

Fig. 3.—Curve showing the rate of swelling of the Ameroid as determined by preoperative and postmortem measurement.

TABLE I. SURVIVAL DATA

DOG NO.	SURVIVAL
520	4 hours
170	1 day
778	1 day
358	1 day
351	2 days
111	2 days
660	3 days
595	3 days
152	3 days
700	5 days
171	6 days
618	6 days
730	7 days
19	7 days
747	8 days
816	8 days
707	9 days
793	10 days
88	10 days

ence rather than the diameter is the effective dimension. The maximum circumferential change was in excess of 4 mm. in 10 days. It can be seen, therefore, that in order to occlude completely the main-stem artery, which has an average diameter of 5 mm., some mild degree of occlusion must be produced by the ligature at the time the wafer is tied in place (Fig. 4). This difficulty was overcome with increasing experience.

A distinct advantage of this method was that an estimate of the degree of occlusion could be made by probing the coronary ostium via the aorta with various sized probes. In the group of animals dying with severe coronary occlusion, 15 had ostia measuring 1.0 mm. or less in diameter; 4 had ostia measuring 2.0 mm. or slightly less. The remaining 10 dogs had widely patent ostia ranging from 2.5 to 5 mm. in diameter; in 1 dog the ligature was distal to the septal branch. All survivors were in the last group.

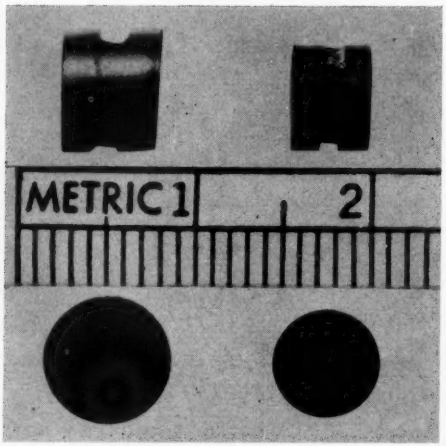

Fig. 4.—Photograph showing Ameroid wafer preoperatively (*right*) and at postmortem, 10 days after operation (*left*).

There was no gross or microscopic evidence of myocardial infarction. In one-half of the animals there was histologic indication of varying degrees of pulmonary edema. Sagittal microscopic sections were made through the artery at the site of the occlusion. These sections do not give an accurate account of the terminal degree of patency of the vessel since the Ameroid and ligature must be removed. They show indentation of the wall of the vessel and varying degrees of pressure necrosis; distal organized thrombi and intimal proliferation are seen occasionally (Figs. 5 and 6).

DISCUSSION

The desired 100 per cent lethality was not obtained with this method of coronary arterial occlusion. However, a subject is produced in which treatment by drug or surgery can be tested, since survivors can be checked accurately for left coronary ostium size by transaortic measurement. More importantly, the

animals produce the picture of an acute coronary insufficiency within a few days and surgical therapy in this acute stage can be put to trial.

This type of acute, major coronary arterial occlusion is lethal and as demonstrated can be associated with ventricular fibrillation. Murray[21] observed that coronary occlusion results in cyanosis, dilation, and asystole in the area of

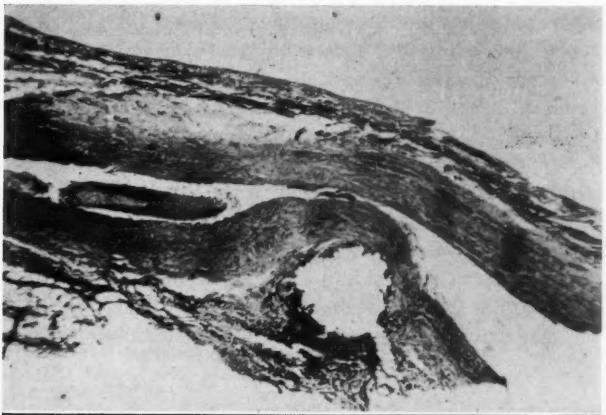

Fig. 5.

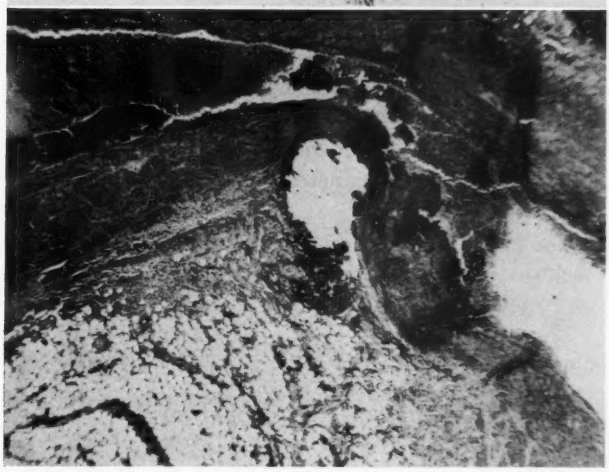

Fig. 6.

Fig. 5.—Sagittal microscopic section of the anterior descending branch and main-stem left coronary artery at the site of occlusion. There is tenting and pressure necrosis of the vessel wall. Organized thrombi can be seen in the anterior descending branch.

Fig. 6.—In addition to the previously noted features, this vessel shows intimal proliferation.

myocardium supplied by the ligated vessel, and he hypothesized that this results in "paradoxical systole," fall in blood pressure, decrease in left ventricular output, and eventual ventricular fibrillation and death. McEachern's studies[22] suggest that the mechanism of ventricular fibrillation may be mediated via the sympathetic nerves from the heart. Beck[23] claims that unequal blood supply

and oxygen potentials result in electrical instability and hence ventricular fibrillation.

Because the dog has intercoronary anastomoses to a greater extent than human beings, the use of this animal has been criticized.[24, 25] However, in this subject the potential protection of many of these anatomoses has been obviated by occlusion at the main stem of the left coronary artery. Survival from occlusion at this point has been rare in other studies. Litvak,[20] in slowly occluding the anterior descending and circumflex rami concomitantly, had all 12 dogs die between 4 and 26 days. Lowenfels[9] was able to occlude slowly the main-stem left coronary artery in 12 dogs and all but 1 died within 1 month. In our series, all the dogs with severe occlusion of the main left coronary artery died within 10 days. These animals showed no gross or microscopic evidence of myocardial infarction and can be compared with the group of patients who die soon after an acute episode of coronary insufficiency. Studies are now in progress challenging the usefulness of arterial grafts from the aorta to the circumflex or anterior descending ramus with this method of occlusion. If this is successful, then staged therapy in the post-occlusion period will be attempted. Comparison can also be made with drug-treated, sympathectomized, and decoagulated subjects.

CONCLUSIONS

1. Occlusion of the main-stem left coronary artery has been produced in the awake, closed-chest dog with a simple ligature encompassing the artery and an absorbing plastic wafer (Ameroid).

2. Nearly complete occlusion of the artery results in the death of the dogs in less than 10 days. This is associated with ventricular arrhythmias, but no histologic evidence of infarction.

3. Lethal occlusion was obtained in 19 of 29 animals in this initial series. Degree of occlusion can be measured transaortically and the surviving group can be separated readily with this indicator of failure to occlude the vessel.

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AUTOTRANSPLANTATION OF THE PULMONIC VALVE INTO THE AORTA

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THE SEARCH continues for the ideal aortic valve replacement. Although plastic valves have provided successful palliation in some cases, their usefulness remains limited. Aortic valve homografts were tried experimentally by Hufnagel[1] and by Lam and co-workers[2] who found that normal function of the cusps did not persist. Murray[3] obtained a more favorable impression of valve homografts from his experimental work and reported their use in a few patients with aortic insufficiency. However, collective clinical and experimental experience with vascular homografts suggests that they will ultimately prove unsatisfactory. The disadvantages of synthetic and homograft valves make the use of a valve autograft an attractive alternative. Several reports[4-10] in the literature have indicated that pulmonic valvular insufficiency may be unusually well tolerated; for this reason the pulmonic valve was selected for investigation as an autogenous transplant.

The purpose of this report is to describe an experimental technique for excision of the intact pulmonic valve and its transplantation into the descending thoracic aorta of the dog. This transplantation is accomplished during total cardiopulmonary bypass utilizing anoxic cardioplegia and selective hypothermia of the heart.

METHOD

Healthy adult mongrel dogs, weighing from 13 to 28 kilograms, were anesthetized with intravenous sodium pentobarbital given in a dosage of 30 mg. per kilogram. One hundred per cent oxygen was administered through a cuffed endotracheal tube with an electronic respirator. Each dog was placed on the right side, and a thoracotomy was performed through the left fourth interspace. Pleural reflections in the anterior mediastinum were divided to allow access to the azygos vein and superior vena cava. The azygos vein was ligated and tapes were passed about the extra-pericardial portions of the superior and inferior

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venae cavae. The latter vessel was approached through the pleural reflection inferior to the pericardial sac and anterior to the phrenic nerve. The pericardium was then opened longitudinally anterior to the phrenic nerve, and its edges were sutured to the muscular borders of the incision, thus creating a pericardial well for the heart and eliminating pulmonary tissue from the operative field.

Following the administration of heparin in a dosage of 2 mg. per kilogram, the superior vena cava was cannulated by passing a catheter through the external jugular vein so that the tip lay just proximal to the occluding tape. The inferior vena cava was cannulated through the right atrial appendage. Insertion of the catheter was facilitated by momentarily rotating the heart to the left.

A rotating disc oxygenator* was utilized in all experiments for total cardiopulmonary bypass. A femoral artery and a carotid artery were cannulated and connected by means of a Y adapter to the arterial limb from the oxygenator. Venous return from the venae cavae was collected in a reservoir by gravity flow and supplied to the oxygenator by a rotary pump. During resection of the pulmonic valve, the heart was rendered quiet and dry by occluding the aorta and pulmonary artery with a cloth-covered Satinsky clamp passed through the transverse sinus. Throughout the period of cardiac anoxia, the pericardial well was perfused with a drip of cold isotonic saline, as described in a previous publication,[11] to provide selective hypothermia of the heart. The left heart was decompressed by a left atrial catheter which diverted bronchial drainage into the venous reservoir. Blood pressure was continuously monitored with a cannula in the contralateral femoral artery. Fig. 1 depicts schematically the bypass technique.

Following the institution of total cardiopulmonary bypass, the aorta and pulmonary artery were occluded. The pulmonary artery was immediately transected just distal to the valve commissures and any blood remaining in the heart was removed. A small transverse incision was made in the anterior wall of the right ventricular outflow tract a few millimeters proximal to the margin of the anterior cusp. The incision of the myocardium along the cusp borders was then completed under direct vision and the annulus was dissected free of the aorta resulting in removal of the valve intact. Special care was taken to avoid injury to the very adherent aorta and to the anterior descending branch of the left coronary artery which courses in close proximity to the pulmonic annulus. After resection of the valve, the aorta was momentarily unclamped to allow identification of bleeding vessels in the cut myocardium and areolar tissue which surrounded the excised segment of the pulmonary artery. These were clamped and ligated. Any injury to the aorta was also identified and sutured at this time.

Despite the demonstration in early experiments that survival was possible with re-anastomosis of the pulmonary artery to the right ventricular outflow tract, it was subsequently decided that a short length of aortic homograft would

*Kay-Cross Oxygenator, Pemco Inc., Cleveland, Ohio.

facilitate restoration of continuity between the right ventricle and pulmonary artery without tension or angulation. Accordingly, a fresh segment of ascending aorta was obtained from a donor animal before each experiment. The homografts averaged 2 cm. in length and the slightly curved shape seemed appropriate for joining the right ventricle and pulmonary artery. Following insertion of the homograft, the aortic clamp was removed and the cold saline drip discontinued.

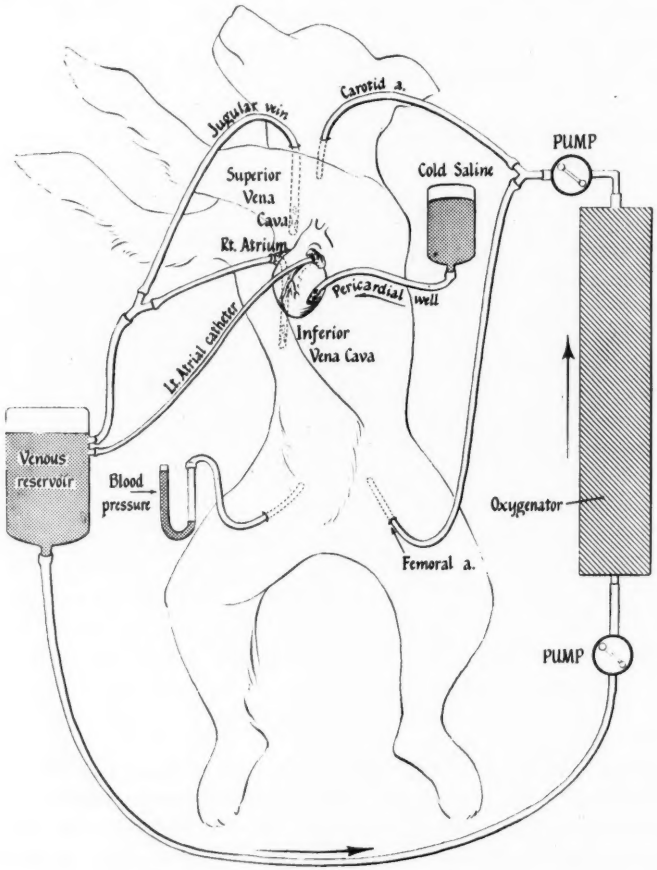

Fig. 1.—Schematic drawing of the heart-lung bypass. The cold saline drip perfuses the pericardial well providing hypothermia of the heart. The left atrial catheter diverts bronchial flow during aortic occlusion.

During the anoxic period, ventricular fibrillation inevitably occurred about 3 to 5 minutes following aortic occlusion; however, the selective hypothermia rendered the fibrillating heart almost motionless. Following aortic disocclusion the cardiac temperature returned toward normal and the fibrillation became

more vigorous. At this point, gentle massage was administered, followed by electric shock defibrillation. In a few instances where defibrillation could not be effected with one or two shocks, a small amount of epinephrine was delivered into the left ventricle to induce coronary vasodilatation. Following this maneuver defibrillation was invariably accomplished.

The second part of the operative procedure was carried out during continued perfusion through the carotid and femoral arteries. The descending aorta was dissected free from its bed for a few centimeters distal to the left subclavian artery and divided between two Potts clamps. The excised pulmonic

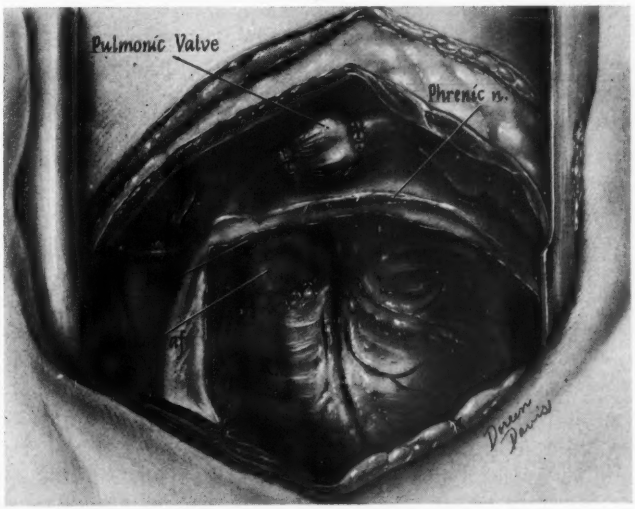

Fig. 2.—The completed transplantation showing the pulmonic valve in the descending aorta and a small segment of arterial homograft restoring continuity between the right ventricle and pulmonary artery.

valve was then interposed between the cut ends of the aorta using a continuous suture technique reinforced where necessary by interrupted sutures. On completion of these anastomoses, aortic clamps were removed and perfusion was gradually terminated. Protamine sulfate in a dosage of approximately 3 mg. per kilogram was administered slowly over several minutes. When bleeding seemed satisfactorily controlled, pleural reflections were sutured over the autograft and the chest was closed in layers. Two chest catheters were attached to suction apparatus for several hours postoperatively, and blood replacement was given when necessary. Penicillin and streptomycin were administered over a 5-day period. Fig. 2 illustrates the completed transplantation of the pulmonic valve.

RESULTS

Thirty dogs were subjected to this operative procedure. The mortality rate was high and uncontrollable bleeding accounted for most of the deaths.

It was interesting to note that recovery of the heart from the anoxic period was usually prompt despite absence of the pulmonic valve.

Bleeding came primarily from two sources. During dissection of the pulmonary artery and valve with the aorta occluded, bleeding points were not easily identified. In addition, damage to the root of the aorta where it is fused to the pulmonic annulus was often undetected. Once the segment of homograft was inserted and the aorta unclamped, it was almost impossible to expose the bleeding vessels. The procedure was then changed to include momentary disocclusion of the aorta following excision of the pulmonic valve and annulus. This allowed the bleeding points to be identified and ligated. This momentary unclamping of the aorta at about the 10-minute point in the anoxic period may have served to supply a small amount of oxygenated blood to the myocardium, although this interval of coronary flow was necessarily very brief to avoid excessive blood loss.

The second troublesome source of hemorrhage was the anastomosis between the transplanted valve and the aorta. Technical difficulty resulted from the disparity in diameter, especially between the proximal or myocardial end of the graft and the descending aorta. In addition, the thinner substance of the graft under systemic pressure tended to balloon out between sutures and provide sources of bleeding. It was soon learned that meticulous placement of sutures was necessary to effect a satisfactory anastomosis. This necessitated long periods of perfusion which seemed uniformly well tolerated. Clotting abnormalities were not apparent in those animals surviving operation.

Another fatal complication of earlier procedures was occlusion of the left anterior descending coronary artery by extensive suturing of the dissected tissues to obtain hemostasis following excision of the pulmonic valve. The subsequent technique of temporary disocclusion of the aorta allowed more precise ligation of bleeding vessels, and the complication of coronary occlusion was usually avoided.

The periods of cardiac anoxia, interrupted only by the momentary unclamping at approximately the 10- or 15-minute point, extended for 30 to 57 minutes. The animals were easily defibrillated and good cardiac action was obtained in all except in the few which had suffered coronary ligation. If subsequent death occurred, it was invariably due to uncontrollable bleeding. In comparison with prior series of animals subjected to comparable periods of anoxia under selective hypothermia,[11] the complete pulmonic valvular insufficiency did not appear to provide an additional burden to the heart during recovery. It was noted that a small area of myocardium about 1 cm. in width adjacent to the anastomosis between the homograft and the right ventricular outflow tract on the anterior surface was frequently infarcted, apparently due to section of a branch from the anterior descending artery. This area has produced no adverse effects.

Table I lists the dogs surviving the operation. The first animal underwent only resection of the pulmonic valve with re-anastomosis of the pulmonary artery and right ventricular outflow tract. When sacrificed at 5 weeks, he

appeared well and the heart was of normal size. Three dogs died within the first 2 weeks from aortic rupture at the suture line. This was to some extent anticipated as extensive suturing was often necessary to control bleeding and this may have resulted in necrosis, particularly at the myocardial end of the graft.

TABLE I. ANIMALS SURVIVING OPERATION

	WEIGHT (KG.)	DURATION OF CARDIAC ANOXIA (MIN.)	TOTAL PERFUSION TIME (MIN.)	RESULT
1	16.0	32	56	Sacrificed at 5 weeks
2	18.3	41	85	Surviving and well
3	22.3	38	76	Surviving and well
4	15.0	41	97	Died fifth postoperative day, rupture of aorta
5	22.3	38	90	Died at 2 weeks, large pericardial effusion
6	20.0	46	106	Surviving and well
7	17.0	40	90	Died at 3 weeks, large pericardial effusion; transplanted valve appeared normal
8	16.0	43	92	Died ninth postoperative day, rupture of aorta
9	15.1	34	92	Died ninth postoperative day, rupture of aorta; infection at site of rupture
10	27.8	47	114	Died tenth postoperative day, empyema
11	18.0	39	93	Died first postoperative day, hemothorax and atelectasis
12	22.0	42	86	Surviving and well
13	21.2	47	99	Surviving and well
14	20.0	39	95	Surviving and well

The surviving animals appear to be normally active and without signs of heart failure. They will be the subject of further study to determine the ultimate fate of the autografted valves.

DISCUSSION

The question of how essential the pulmonic valve is to normal circulatory function remains incompletely answered. Although isolated pulmonic insufficiency is rarely encountered clinically, the increasing frequency of surgery for congenital pulmonic stenosis raises a practical question concerning the risk of surgically producing pulmonic valvular insufficiency. Accordingly, several investigators have produced varying degrees of experimental, pulmonic insufficiency in attempting to elucidate the significance of this lesion.

Ellison[4] and Ratcliffe[5] and their associates subjected dogs to complete excision of the pulmonic cusps. Their most striking observation was that the animals remained asymptomatic even though some were subjected to regular exercise. The former group presented cardiac catheterization studies which confirmed the presence of free pulmonic regurgitation but demonstrated in all animals normal cardiac output and normal end-diastolic pressures in the right

ventricle. Pathologic examination of the animals showed in some instances right ventricular dilatation and hypertrophy after 12 months' observation. Kay and Thomas[6] performed complete removal of pulmonic valve cusps in a group of dogs and subsequently described right ventricular dilatation in all specimens. The dilatation was of marked degree in 4 animals and 1 animal was reported as having died of right heart failure. One might speculate that this animal may have been subjected to some additional acute stress such as pulmonary infection or infarction.

Clinical reports of organic pulmonic valvular insufficiency in human patients have likewise illustrated that such a lesion may exist for prolonged periods without producing symptoms. Ford and his co-workers[7] reviewed cases of bicuspid pulmonic valve, in which pulmonic insufficiency is a prominent feature, and concluded that the lesion is compatible with a good prognosis and is seldom if ever the cause of death. Campeau and associates[8] described a 32-year-old woman with congenital absence of the pulmonic cusps and a ventricular septal defect who was asymptomatic until the age of 30. Lendrum and Shaffer[9] presented catheterization findings characteristic of free pulmonic regurgitation in a 16-year-old boy who was asymptomatic although leading an active life including competitive athletics. Ehrenhaft,[10] in discussing the experimental work of Ellison and his associates, described a 14-year-old boy who at operation was found to have no pulmonic cusps. He had a slightly decreased exercise tolerance, but the lesion was well tolerated for 10 years.

The pulmonic valve has therefore been selected for investigation as an autogenous transplant. The question of ultimate tissue viability remains to be answered, and it is hoped that the experimental preparation herein described will subsequently contribute this information.

The feature of a homograft segment in the pulmonary artery has previously been reported by Robinson and his colleagues[13] who found ulcerations of the intima, calcifications in the wall of the graft, and, frequently, small adherent thrombi. The ultimate fate of such grafts has not yet been reported and it is likely that the homograft will remain patent long enough to serve its purpose in this investigation.

The pathologic and physiologic findings in the surviving animals will be the subject of a subsequent report. To date they have not shown any signs of disability despite absence of the pulmonic valve. Further experiments are planned to examine the behavior of pulmonic valve autotransplants used to replace the mitral or tricuspid valve.

SUMMARY

A technique has been described for autotransplantation of the intact pulmonic valve into the descending thoracic aorta of the dog utilizing cardiopulmonary bypass and selective hypothermia of the heart. The pulmonic valve was selected for transplantation as there exists both clinical and experimental evidence that complete pulmonic insufficiency may be well tolerated for prolonged periods.

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HEART BLOCK FOLLOWING INTRACARDIAC SURGERY: LOCALIZATION OF CONDUCTION TISSUE INJURY

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HEART BLOCK remains a distressingly frequent complication of intracardiac repair of certain congenital anomalies, particularly ventricular septal defect occurring as an isolated lesion or in such complexes as tetralogy of Fallot and common atrioventricular canal. Several recent studies have demonstrated the anatomic relationships of conduction tissue to various intracardiac defects,[1-4] but no studies have been reported which show the site of injury following operation. The present report deals with the localization of injury to conduction tissue in patients whose intracardiac procedures were complicated by complete heart block.

MATERIAL

Specimens were obtained from 3 patients who had undergone open-heart repair of congenital cardiac defects. In each case complete heart block was observed during or immediately following suturing of the defect, and in each patient heart block was considered to be the primary cause of death.

CASE REPORTS

CASE 1.—The patient was a 5-year-old child who was found to have a heart murmur at 2 months of age, cardiomegaly at 6 months of age, and cyanosis at 2½ years. Cardiac catheterization performed on two occasions was interpreted as demonstrating an atrioventricular canal. At operation, on Oct. 31, 1958, a persistent atrioventricular canal with a large interatrial component, a small ventricular septal defect, and a cleft of the mitral valve were found. These defects were repaired without cardiac arrest. After 33 minutes of perfusion the patient was taken off the pump-oxygenator and heart block was noted. Her immediate postoperative course was complicated by hypotension and respiratory insufficiency. Despite supportive measures her condition deteriorated and she died 20 hours following operation.

CASE 2.—This patient was a 4-year-old child with tetralogy of Fallot who underwent open-heart surgery on Nov. 6, 1958. With the patient on cardiopulmonary bypass, cardiac arrest was induced with potassium citrate. The right ventricle was opened and the ventricular septal defect repaired. An Ivalon patch was placed in the right ventricular outflow tract, extending into the pulmonary artery. After release of the aortic clamp and massage,

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a cardiac beat was observed but the electrocardiogram showed complete heart block. Postoperatively the patient remained unresponsive and cyanotic. Respiratory arrest occurred 3 hours after the conclusion of surgery and was followed by cardiac arrest. Despite emergency thoracotomy and massage, the heartbeat could not be re-established.

CASE 3.—On cardiac catheterization of this 7-year-old child, a ventricular septal defect, with no evidence of right-to-left shunt, and a pulmonary artery pressure of 85/40 were demonstrated. On March 5, 1959, she underwent repair of the interventricular septal defect. At the end of the repair, complete heart block was noted on the electrocardiogram and electrodes were placed in the heart and subcutaneous tissues. Following operation her immediate course was satisfactory and the heart rate was maintained by an electronic pacemaker. However, during the subsequent 2 weeks her condition deteriorated despite maintenance of the heart rate with the pacemaker. She remained in complete heart block and showed evidence of progressive deterioration, with death on the seventeenth postoperative day.

METHODS

Each heart was examined and photographed with the area of repair undisturbed. Sutures were then removed and the specimens photographed with the defects open. From each specimen a block of tissue was removed which completely encircled the defect, including the coronary sinus, the inferior portions of the atrial walls and interatrial septum, and the superior portion of the ventricular walls and interventricular septum. This block was sectioned in a direction corresponding approximately to the anterior-posterior plane of the body. Sections were stained alternately with hematoxylin and eosin and Masson's triple stain. Graphic reconstructions of the conduction system were made following histologic study of the slides.

OBSERVATIONS

Case 1.—This specimen showed a common atrioventricular canal. A common atrioventricular valve extended through the defect, and there was a cleft in the mitral portion of the valve. Postmortem examination showed satisfactory repair of the mitral cleft and complete closure of the atrial and ventricular septal defects (Fig. 1). Microscopic study showed the atrioventricular bundle lying subendocardially along the posterior-inferior margin of the ventricular portion of the atrioventricular canal. Suture holes and extensive hemorrhage were observed in the conduction bundle in this area (Figs. 2 and 3).

Case 2.—This specimen showed the characteristic findings of the tetralogy of Fallot. The pulmonary outflow tract had been widened by the insertion of a diamond-shaped Ivalon patch which extended into the pulmonary artery. The ventricular septal defect (Fig. 4) had been incompletely closed, a 4 mm. defect persisting in the superior portion. Microscopic study showed extensive hemorrhage in and around the conduction bundle (Fig. 5), in the area of the posterior-inferior margin of the defect (Fig. 6).

Case 3.—This specimen showed a ventricular septal defect at the origin of the outflow tract of the right ventricle, anterior to the septal leaflet of the tricuspid valve. The defect had been closed satisfactorily (Fig. 7). Microscopic sections demonstrated suture tracts and hemorrhage in the conduction bundle (Fig. 8) along the posterior-inferior rim of the defect (Fig. 9).

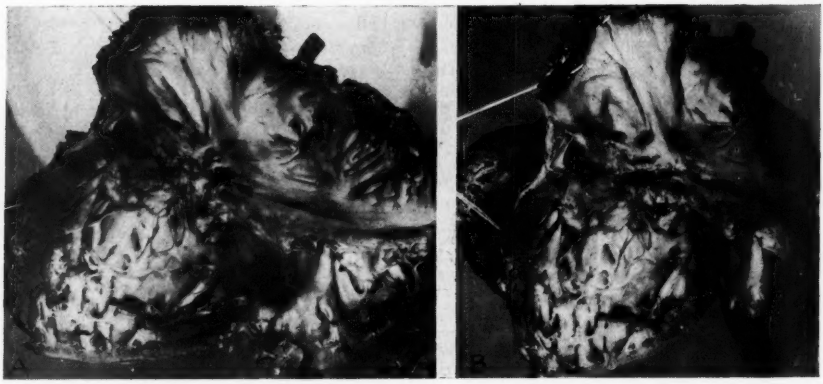

Fig. 1.—Case 1. *A*, Atrioventricular canal, viewed from the right side of the heart. The right atrium lies above and the right ventricle below. The coronary sinus is seen adjacent to the repaired defect. *B*, The sutures have been removed, disclosing the atrioventricular defect.

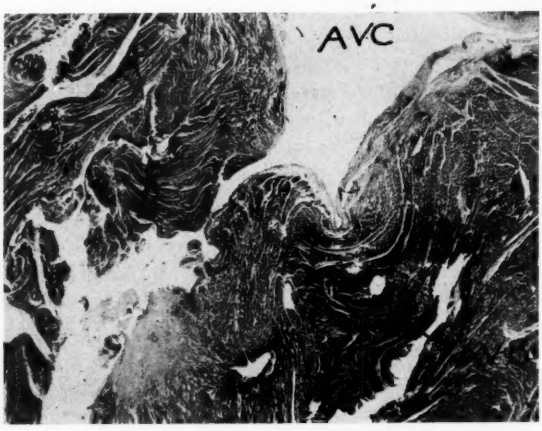

Fig. 2.—Case 1. Photomicrograph showing the atrioventricular canal (*AVC*) and the atrioventricular bundle (*AVB*). Suture holes are seen in the bundle, and extensive hemorrhage is apparent in the area of the conduction tissue.

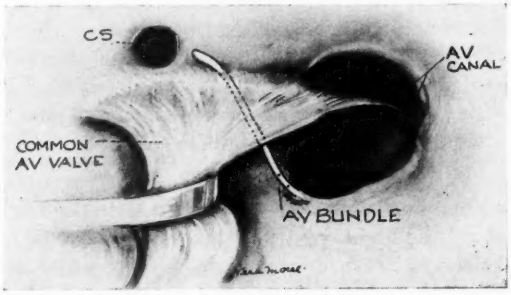

Fig. 3.—Case 1. Drawing of the specimen of atrioventricular canal, showing the relationship of the atrioventricular bundle to the A-V canal. The site of injury of the A-V bundle lay along the posterior-inferior aspect of the defect.

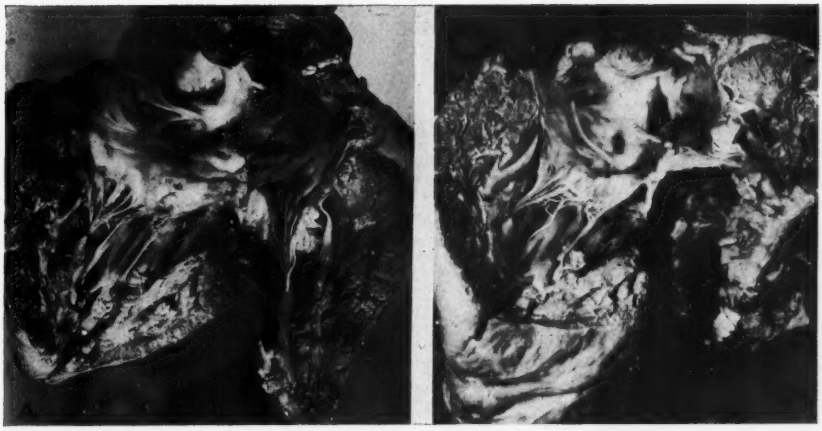

Fig. 4.—Case 2. *A*, Tetralogy of Fallot viewed from the right side of the heart. Sutures are seen in the ventricular septal defect. The outflow tract is not shown. *B*, The sutures have been removed and the point of the hemostat shows the location of the ventricular septal defect.

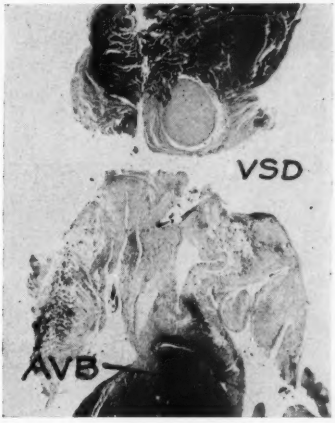

Fig. 5.—Case 2. Photomicrograph showing the ventricular septal defect and the atrioventricular bundle. There is extensive hemorrhage both in the atrioventricular bundle and in the surrounding ventricular septum.

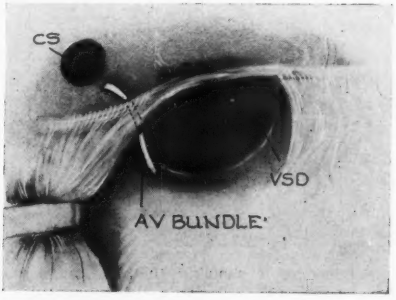

Fig. 6.—Case 2. Drawing showing the relationship of the atrioventricular bundle to the ventricular septal defect in the tetralogy of Fallot. The area of hemorrhage occurred along the posterior-inferior aspect of the defect.

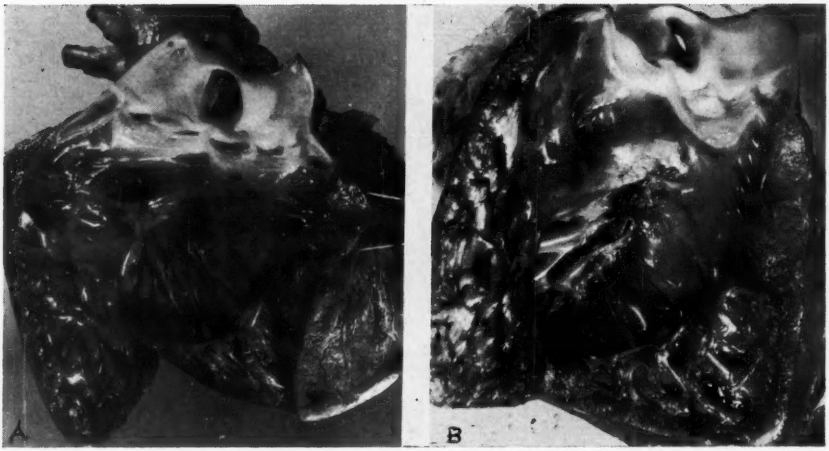

Fig. 7.—Case 3. *A*, Photograph of the case of ventricular septal defect showing the sutures in place. The defect lies at the origin of the outflow tract. The pulmonary valve and artery are seen above the defect. *B*, The sutures have been removed, demonstrating the ventricular septal defect.

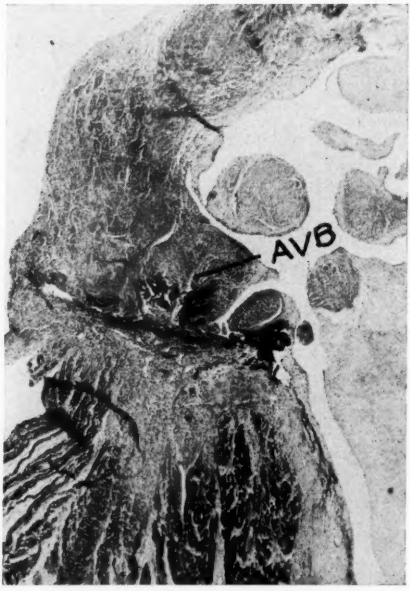

Fig. 8.—Case 3. Photomicrograph showing the atrioventricular bundle above the top of the ventricular septum. A suture tract is seen through the bundle and there is extensive hemorrhage within the bundle itself.

DISCUSSION

The conduction system frequently lies in a vulnerable position in relation to certain intracardiac defects. In the majority of cases of ventricular septal defect occurring as an isolated lesion or in such complexes as tetralogy of Fallot and common atrioventricular canal, the atrioventricular bundle lies subendocardially along the posterior-inferior margin of the ventricular defect.

In the 3 cases included in the present report, heart block occurred following the repair of the cardiac defects in each. In one, the ventricular septal defect occurred as an isolated lesion. In the other 2 cases the ventricular defect was associated with the tetralogy of Fallot in one and with an atrioventricular canal in the other. Yet the site of injury of the conduction system was common to all: the posterior-inferior margin of the septal defect. In each case extensive hemorrhage was noted in and around the bundle in this area, and suture tracts through the bundle were noted in 2.

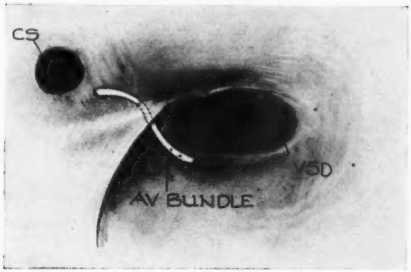

Fig. 9.—Case 3. Drawing of the specimen with a ventricular septal defect. The site of injury of the conduction bundle is along the posterior-inferior aspect of the ventricular septal defect.

SUMMARY

1. Injuries to the conduction system were studied in 3 cases of complete heart block complicating repair of intracardiac defects.

2. In one case the ventricular septal defect occurred as an isolated lesion. In the other cases the ventricular septal defects were associated with the tetralogy of Fallot in one and with a common atrioventricular canal in another.

3. Extensive hemorrhage in and around the atrioventricular bundle occurred in each case. Suture tracts through the bundle were observed in 2.

4. In all cases the site of injury was located along the posterior-inferior rim of the ventricular septal defect.

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A NEW INSTRUMENT TO MAINTAIN PATENCY OF DRAINAGE TUBES

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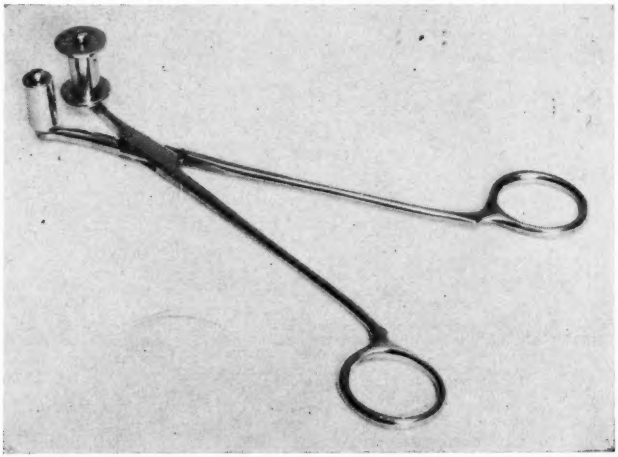
WHENEVER DRAINS or tubes are used after thoracic surgery, it is important to keep them well functioning and free of pus and blood clots which may be difficult to milk down the tubes.



To facilitate milking down the tubes, we have invented an instrument* with "rolls," as shown in the accompanying figure. It has been used in the Thoracic-Surgical Department at Øresunds Hospital for several months to the great satisfaction of both nurses and doctors.

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*The clamp is manufactured by Simonsen and Weel, Copenhagen, Denmark.